ANNALS
OF
OTOLOGY, RHINOLOGY
AND
LARYNGOLOGY

VOL. 47 JUNE, 1938 No. 2

XXIV

THE BASE OF THE SKULL, WITH PARTICULAR REFERENCE TO FRACTURES*

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The base of the skull is the "gateway" to the cranium, for through it all the important blood vessels and nerves must pass going to and from the brain case. The floor of the cranial cavity is not in a horizontal plane, but placed at an angle of about thirty degrees with such a plane, tilting upward in front. The floor of the cerebellar fossa is about two inches lower than the floor of the anterior fossa, and about one inch lower than the floor of the middle fossa (Fig. 1). The openings through the base of the skull are thus directed either forward or downward. The firm attachment of the dura over the entire inner surface of the base is an important consideration. It is very readily torn along fracture lines and its firm attachment accounts for the infrequency of extra-dural hemorrhages in this region.

Fractures usually occur through points and lines of weakness. In the base of the skull these are across and in line with two or more foramina, along the sutures and through the thin bony plates in the floor of the cranial fossæ. These plates are often very thin by reason of pneumatization within the various bones. This is particularly true with the sphenoid bone and less often with the orbital plate of the frontal bone in instances where the frontal sinus extends well to

*Presented before the Western Section of the American Laryngological, Rhinological and Otological Society, Santa Barbara, January 30, 1938.

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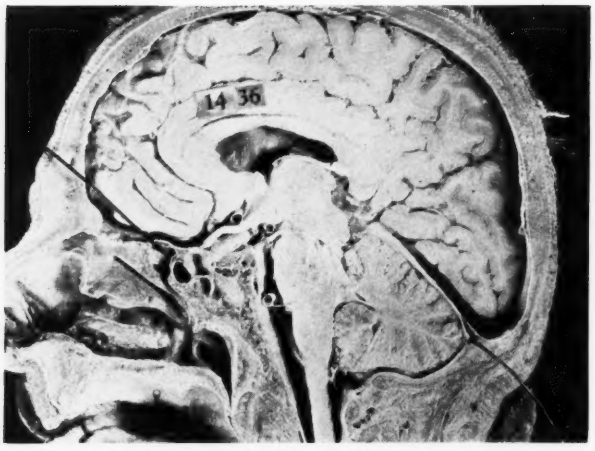

Fig. 1. Mid-saggital section of the head to show the plane of the floor of the posterior fossa with reference to the plane of the floor of the anterior fossa. Note the basi-sphenoid is not pneumatized, so would act as a buttress in event of fracture.

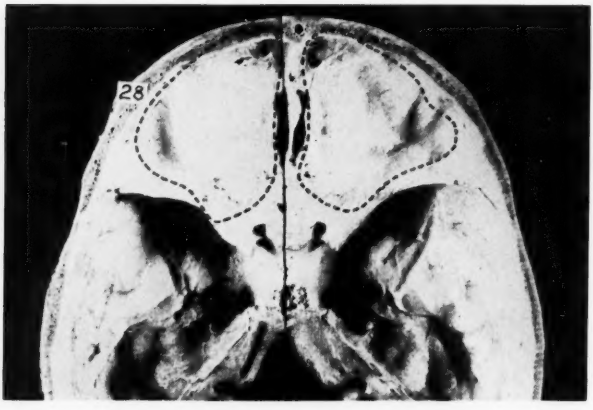

Fig. 2. Base of the skull in which the frontal sinuses extend backward and laterally under almost the entire floor of the anterior fossa. Their extent is indicated by the dotted lines.

the posterior limit (Fig. 2). The ethmoidal cells may also extend into the orbital plate of the frontal bone (Fig. 3). Pneumatization is so variable as to extent and direction that it becomes a very uncertain and hazardous factor. Bones in which air cells are normally found may be pneumatized to the limit in any or all directions and the process may extend across suture lines into adjoining bones if fusion of the suture is complete early in development, as in the temporal bone. It is often demonstrable across the spheno-occipital suture and follows early fusion with the formation of diploic structure. Occasionally the process extends beyond the limit of the bone, forming a diverticulum of the air cell. This has been observed by the writer in four instances in connection with the extension of the sphenoidal sinus into the lesser wing on one or both sides of the optic canal. The pouched air cell extended not only through the bone but through the dura and lay free in the subdural space.

The sphenoid bone is the "keystone" of the skull; situated in the middle of the base, it enters into the formation of all three cranial fossæ, and articulates with all the bones entering into their formation. The body rises high in the midline and divides the middle fossa into right and left compartments. The pituitary fossa and the grooves on either side for the internal carotid artery are very vulnerable areas, for here the bone may be paper-thin or wanting entirely at points along the carotid groove. The optic sulcus and canals may likewise, by reason of very thin bony walls, present little resistance to forces tending to produce fractures (Fig. 4). The sphenoidal sinus may extend downward into the pterygoid process or laterally to the limit of the greater wing. In 129 specimens examined, the average width of the right and left sinus together was one-third of the width of the skull. In one specimen the combined width of the sphenoidal sinuses was 7.5 cm., the width of the head 12.5 cm. The lesser wings may be completely pneumatized by the sphenoidal and ethmoidal cells, the optic canal being almost completely surrounded by air spaces (Fig. 5). In thirty-five per cent of specimens examined, the sphenoidal sinus extended backward to the posterior fossa with a bony wall of 3 mm., or less in thickness, and many showed pneumatization into the posterior clinoid processes (Fig. 6). As indicated above, the frontal sinus and ethmoidal cells may extend backward and outward to cover the entire orbit, thus bringing all the floor of the anterior fossa into intimate relation with outside influences (Figs. 2 and 3). The temporal bone may be pneumatic to the limit of the apex and in the anteroposterior direction cells may extend from back of the knee to the forward limit of the squama near the front boundary of the middle fossa (Fig. 7).

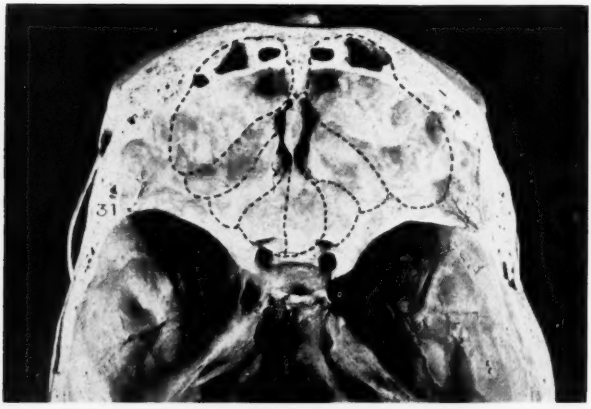

Fig. 3. Base of the skull with the extent of the frontal sinuses and of the posterior ethmoidal cells indicated by the dotted lines.

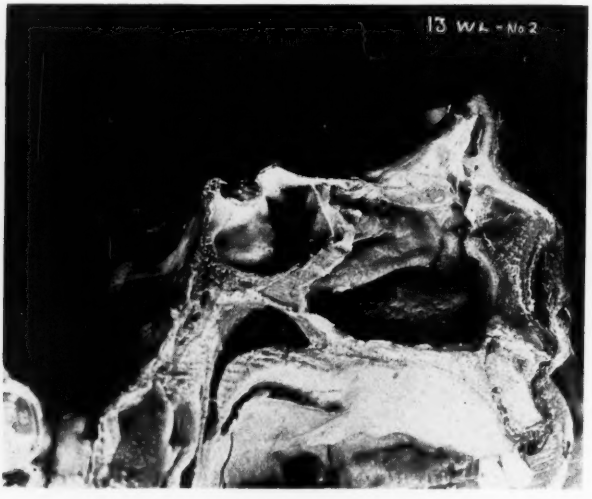

Fig. 4. Mid-saggital section of the head showing the extension of the sphenoidal sinus into the lesser wing of the sphenoid surrounding the optic canal.

The possible areas of pneumatization then are the whole of the anterior fossa, practically all of the middle and a portion of the posterior fossa; the latter from three courses, namely, mastoid cells back of the knee and those along the sigmoid sinus to the jugular bulb, cells in the posterior part of the pyramid to the apex, and sphenoidal sinus extension into the basilar process.

According to the excellent analysis of Rawling,[1] the usual sites of the impact of forces producing fractures and the usual lines of fractures are as follows (Fig. 8):

1. Site—the midfrontal region. Fracture through the cribriform plate of the ethmoid, posterior ethmoidal cells, sphenoidal sinus, pituitary fossa, carotid canal, along petro-occipital suture, jugular foramen and groove for sigmoid sinus. In line of such a fracture would be found:

A. Nerves.

Olfactory, vidian, oculomotor, trochlear, abducens, ophthalmic and maxillary divisions of the trigeminal, glossopharyngeal, vagus and spinal accessory.

B. Blood Vessels.

Anterior and posterior ethmoidals, sphenopalatine artery, cavernous sinus, internal carotid, inferior petrosal sinus, petro-occipital sinus, jugular bulb and sigmoid sinus.

2. Site—external angular process or lateral frontal region. Fracture through the orbital plate of the frontal bone, posterior optic foramen, pituitary fossa, carotid canal, eustachian tube, tympanum and external auditory canal. In line of such a fracture would be found:

A. Nerves.

Optic, oculomotor, trochlear, abducens, vidian, Gasserian ganglion, great superficial petrosal, facial and the carotid sympathetic plexus.

B. Blood Vessels.

Ophthalmic artery and veins, internal carotid, cavernous sinus and middle meningeal artery.

3. Site—external ear. Fracture through the roof and floor of the external auditory meatus, tympanic membrane, tegmen tympani, eustachian tube, carotid canal and body of the sphenoid. In line of such a fracture would be found:

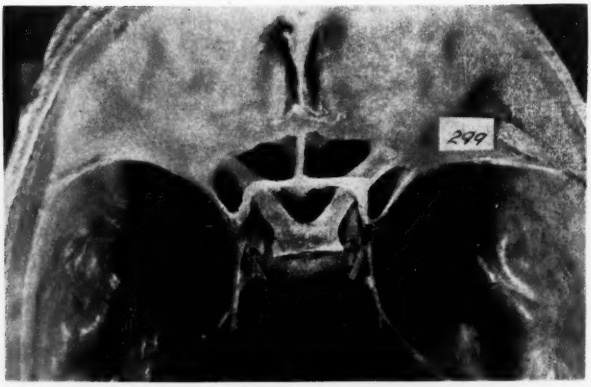

Fig. 5. Dissection showing posterior ethmoidal cells and sphenoidal sinuses from above. Note the optic canals are thin bony tubes surrounded by air cells and that the anterior clinoid processes are completely pneumatized, the left by a posterior ethmoidal cell and the right by the extension of the right sphenoidal sinus under the optic canal. The internal carotid artery makes its forward bend in this cell and has no bony protection over a considerable area.

Fig. 6. Mid-saggital section of the head showing extensive pneumatization of the sphenoid. Note the thin posterior wall and its relation to the cerebellar fossa. The bulge on the lateral wall is dehiscent over the internal carotid artery.

A. Nerves.

Facial, great superficial petrosal, gasserian ganglion, maxillary, mandibular, carotid sympathetic plexus, abducens, and vidian.

B. Blood Vessels.

Tympanic plexus, internal carotid, middle meningeal and cavernous sinus.

4. Site—mastoid region. Fracture through the mastoid cells, jugular foramen, along the petro-occipital suture, and then through the body of the sphenoid. In line of such a fracture would be found:

A. Nerves.

Glossopharyngeal, vagus, spinal accessory, abducens, vidian, and carotid sympathetic plexus.

B. Blood Vessels.

Mastoid emissary vein, sigmoid sinus, jugular bulb, inferior petrosal sinus, petro-occipital sinus, cavernous sinus and internal carotid artery.

5. Site—occipital region. Fracture through the thin floor of the cerebellar fossa across the foramen magnum to condylar foramen just back of the condyle, the jugular foramen, then through the pyramid of the temporal bone just lateral to the internal auditory meatus. In line of such a fracture would be found:

A. Nerves.

Glossopharyngeal, vagus, spinal accessory, auditory and facial.

B. Blood Vessels.

Lateral sinus, vertebral artery and veins, condylar emissary vein, jugular bulb, tympanic vessels and posterior branch of the middle meningeal.

This classification is simple and practical, but it must be remembered that the extent and direction of the fracture line depends upon the strength of the force applied and the size of the object whose impact causes the fracture. Fractures vary from slight linear fractures hardly demonstrable at autopsy, to lines running in every direction throughout the base of the skull. A small object at high speed will produce very little shattering of the bone, whereas, a blow

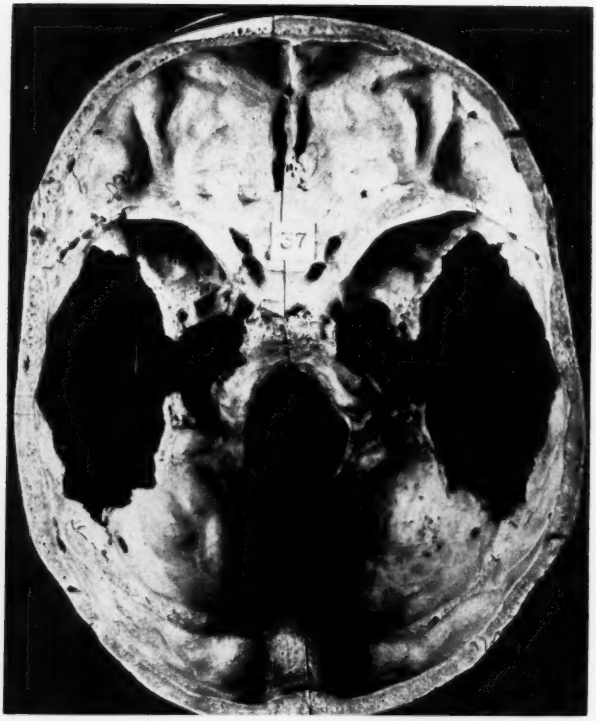

Fig. 7. Base of the skull with the temporal bones removed. Note the extent of the area occupied by them. Air cells may be present in any portion of this bone.

over a wide area tends to cause branching of the fracture lines. The thickness of the bones, the development of diploe and diploic veins, the rigidity of the sutures, as well as the fundamental composition of the osseous structure, all have a bearing upon the direction and extent of the fracture. Whether the process of pneumatization continues throughout life is not known, but there is a very definite tendency for the bony walls of the sinuses to become thinner as age advances. This, with the fact that the bones tend to become brittle, makes the base of the skull very vulnerable in the aged. Occasionally certain sutures (spheno-occipital and frontal) remain open throughout life and must be taken into account in the interpretation of x-ray films. In head injuries one must keep in mind that a fatal blow may occur without producing a fracture, also rather extensive fractures may be present without loss of consciousness; the internal ear may be put out of commission from hemorrhage within the labyrinth without a fracture of the bony capsule. Transverse fractures of the petrous pyramid usually extend through the labyrinth and seldom heal by bony union because the endochondral layer is not endowed with regenerative power.

It is important to note that all fracture lines except those through the posterior fossa tend toward the sphenoid, and if the force is not spent it is deflected by the basi-sphenoid to continue along the anterior border of the petrous pyramid. If the sphenoidal sinus extends backward into the basilar portion, then the line is more liable to extend along the posterior border of the pyramid. The weakest part of the sphenoid is the pneumatized portion, which is often comminuted. It will thus be apparent that a large percentage of basal fractures are compound in that they communicate with the outside by way of some air cell.

The temporal is one of the most complicated and interesting bones of the cranium, in that it contains many important structures, or is intimately related thereto. It is situated at the side and in the base of the skull forming approximately two-thirds of the floor of the middle, and about one-third of the floor of the posterior fossa.

To get a true picture of this arrangement, study a skull with both temporal bones removed (Fig. 7) and note the extent of the area occupied by them; then look at the skull with one temporal bone in place, and compare the two sides, remembering that a large part or all of this area may be pneumatized.

The temporal bone is unique in its connection with adjoining bones. The sutures tend to remain open with fibrous tissue filling

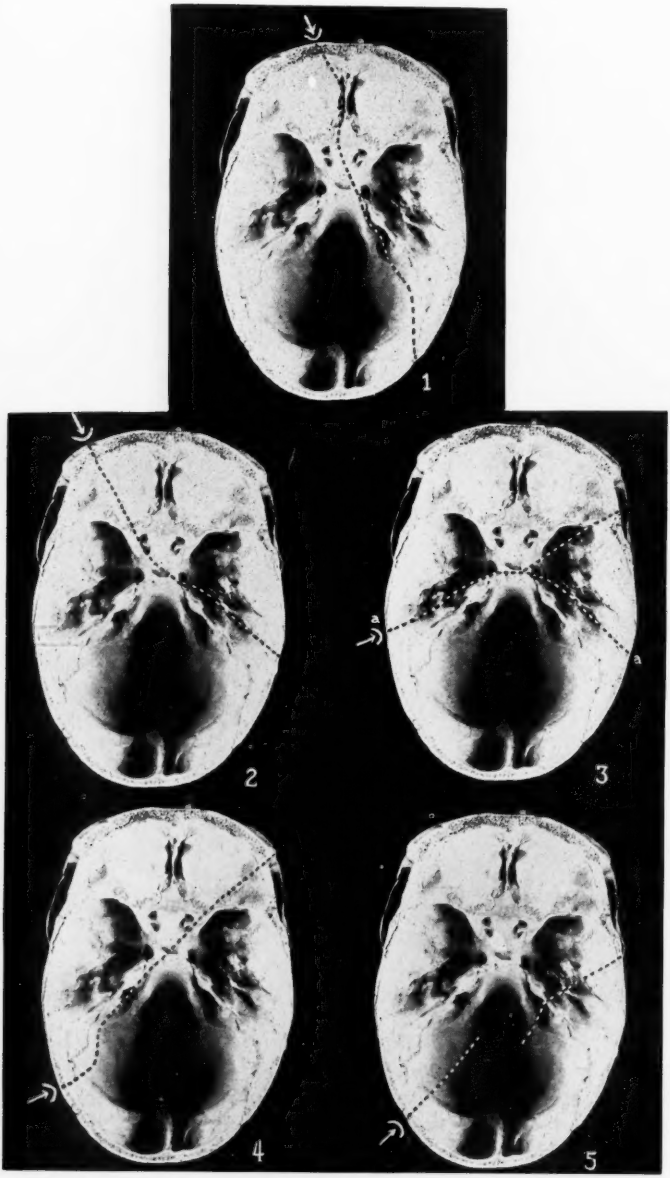

Fig. 8. Lines of fracture through base of the skull (after Rawling). 1. Force applied in mid-frontal region. 2. Force applied in lateral frontal region. 3. Force applied in region of external ear. a-a The "typical basic fracture." 4. Force applied in mastoid region. 5. Force applied in lateral occipital region.

the gaps. The upward forward part of the squamous portion is beveled over a wide surface to articulate with a similar area on the parietal and outer surface of the greater wing of the sphenoid. The outer border of the greater wing in front of the spinous process is also beveled to receive the lower border of the squama. It is also interesting to note that the zygomatic process is beveled in such a way as to rest on top of the temporal process of the malar bone. The petrous apex is loosely attached to the posterior border of the greater wing and the basilar process. By this arrangement one will observe that a certain amount of movement or give from below upward is provided for, thus protecting the hearing apparatus against possible damage from forcible closure of the jaws, or perhaps from shock which might come from a blow on the chin. If the force to the chin be sufficient, it can readily be understood how the whole temporal bone may be displaced upward, or if firmly attached along the sutures, the condyle of the mandible may be driven into the glenoid fossa producing an extensive fracture in the floor of the middle cranial fossa. The mastoid portion of the temporal bone may become fused with the occipital and parietal bones and pneumatic cells may extend into these bones for a considerable distance. Law[2] reports a case in which radiograms demonstrated the extension of such cells on both sides, backward to within about 0.7 mm., of the midline at the occipital protuberance.

In fractures involving the olfactory region, swelling of the nasal mucosa occurs, which results in loss of the sense of smell due to compression of the olfactory nerves. This is of course only temporary, as there is seldom sufficient damage to enough of the fibers as they pass through the tiny foramina in the cribiform plate to cause permanent loss of function.

The optic nerve, due to its long course and unique arrangement within its sheath, may be affected in several different ways. Function may be suspended by hemorrhage within the sheath from rupture of the central artery of the retina, or by pressure from without, due to injury to some of the orbital vessels. Partial or complete avulsion of the optic nerve may follow or accompany fractures of the nasal and maxillary bones. It may be severed or crushed at the posterior optic foramen, or within the optic canal where the bony walls are often very thin.

The oculomotor, trochlear, abducens and the ophthalmic division of the trigeminal may all or any one be involved as they pass through the superior orbital fissure. One or more may be compressed by hemorrhage within the orbit giving rise to pupillary changes, squint, etc.

The gasserian ganglion or any of its three divisions may be involved, giving rise to pain or anesthesia in the frontal region, eye (cornea), cheek or teeth.

The mandibular nerve may be compressed by shattered bone or hemorrhage at the foramen ovale, paresis or paralysis of the buccinator and masseter muscles with neuralgia or numbness of the teeth might follow. If the lingual be injured the chorda tympani which joins it would likely be included, with the result that not only would there be anesthesia of the anterior two-thirds of the tongue, but also a loss of taste in the same region. There would also be a disturbance of salivary secretion from the sublingual and submaxillary glands; herpes of the lip might occur from involvement of the vasomotor portion.

The abducens deserves special consideration for it is one of the nerves most frequently affected by disturbances at the base of the brain. This is due chiefly to the fact that it is surrounded by venous blood spaces throughout most of its long course. When it pierces the dura on the basilar process, it passes through the basilar plexus of veins on its way outward and upward in intimate relation with the inferior petrosal sinus. The two pass through Dorello's canal together. Here the nerve is usually on the lateral side of the sinus, but it may travel on the mesial side, or it may be in the middle of the sinus, with blood on either side as well as above it; in other words, it may be exposed to the blood current on three sides. It then enters the cavernous sinus where it is in immediate relation with the internal carotid and is exposed on all sides to the blood in the cavernous sinus until it enters the orbit. Its relation to osseous structures is very important as it may be compressed from a blow on the side of the head driving the petrous apex against the sphenoid. If it follows the lateral wall of the inferior petrosal sinus, it then lies in a groove in the very tip of the petrous bone. Occasionally it runs through the gap or suture between the apex and the basilar process, thereby being placed in a very vulnerable position. In instances where the basilar process is very wide and well pneumatized the nerve may then cause a bulge into the sphenoidal sinus under the posterior clinoid, and be injured by a comminuting fracture of the sphenoid. At times the petrosphenoidal ligament is absent, then the nerve may pass over the superior border of the apex some distance from possible injury.

In fractures of the middle fossa the facial is more often involved than any other nerve. It is also affected in posterior fossa fractures with a transverse fracture through the pyramid. Here the damage is apt to be complete and permanent, whereas in the longitudinal

fractures of the petrous pyramid it is more likely to be a partial loss of function. In either case the injury is almost always in the region of the genu. If complete, in addition to facial paralysis and ptosis, there would be a loss of the sense of taste on the anterior two-thirds of the tongue as well as disturbed salivary secretion of the submaxillary and sublingual glands by reason of injury to the chorda tympani. The greater superficial petrosal would also suffer with the resultant disturbances in lacrimal and nasal secretion. Herpes of the ear often follows involvement of the vasomotor portion.

As stated above, cessation of function of the auditory nerve may be caused by hemorrhage within the labyrinth without a fracture, in which case there may be partial or complete recovery. In transverse fractures of the pyramid both the internal and middle ear are involved with complete loss of cochlear and vestibular function. In partial involvements, vertigo and tinnitus may be severe and lasting.

The glossopharyngeal, vagus, and spinal accessory, while lying in the line of fracture in many instances, are seldom injured because they are embedded in soft tissue at their exit, in the middle of the jugular foramen between the jugular bulb and the inferior petrosal sinus. If the glossopharyngeal be injured there would be a loss of taste on the posterior part of the tongue with difficulty in swallowing. Injury to the vagus would give rise to hoarseness and a rapid heart, while involvement of the spinal accessory would produce shoulder dropping and inability to turn the head from weakness or paralysis of the trapezius and sternomastoid muscles. If the hypoglossal were involved, the tongue would protrude to the opposite side.

The vidian nerve may, on account of its course through the body of the sphenoid, be alone involved in fractures affecting this region. There would result disturbances in lacrimal and nasal secretion, also deep seated ocular pain may accompany such an injury.

Hemorrhage into the nasal cavity or nasopharynx may escape through the nose and mouth or be swallowed and vomited. Laceration of the nasal mucosa or the lining membrane in the air cells may cause considerable bleeding for a time, and if continuous is probably due to injury of one of the larger vessels. The first in line of injury when the fracture is through the cribriform plate are the ethmoidal arteries, terminal branches of the ophthalmic. The sphenopalatine, terminal branch of the internal maxillary artery, may be torn as it crosses the front wall of the sphenoid. The ophthalmic may be injured in the optic canal and bleed into a posterior ethmoidal cell or into the sphenoidal sinus; likewise the internal carotid or cavernous sinus, as well as the basilar artery and plexus may be injured with

blood escaping into the sphenoidal sinus. It must be remembered, however, that the injured vessel may be on the side opposite to that from which the blood is escaping. In other words, one sphenoidal sinus may extend to the opposite side, bringing both internal carotid or ophthalmic arteries into relation with it, and the injured artery be the one on the opposite side. Blood from the middle meningeal may escape into the nasopharynx by way of the eustachian tube.

In the region of the orbit hemorrhage due to fractures of the base may manifest itself in many ways; ecchymosis in the upper lid towards the inner canthus is always suggestive, especially if it is bilateral. It is quite positive if it comes on several days following the injury. Papilledema, usually bilateral, may be marked within a few hours, due to hemorrhage within the vaginal sheaths from lacerations in the optic canal, or may accompany a rather severe subarachnoid hemorrhage. Bleeding within the orbit may cause acute exophthalmos, or choked disk, but as a rule is unilateral. Both optic nerves may be involved from large hemorrhages in the region of the chiasm. Injury to the internal carotid in the cavernous sinus may produce an arteriovenous aneurism with a resultant pulsating exophthalmos.

Bleeding from the ear may come from the tympanic plexus, sigmoid sinus, superior petrosal sinus, jugular bulb or from the middle meningeal. It is also possible to have an injury of the internal carotid with blood escaping by way of the external meatus. As a rule transverse fractures of the pyramid do not involve the tympanic membrane; therefore hemorrhage from the ear in head injuries usually means a horizontal fracture of the pyramid, and is almost pathognomonic of middle fossa fractures.

Severe comminuting fractures involving the tegmen tympani and the cribriform plate of the ethmoid may give rise to the escape of cerebrospinal fluid, which means the dural and arachnoidal prolongations along the nerves have been ruptured. This opens the base of the brain to possible infection. Brain matter may also escape from these areas, but does not necessarily indicate a hopeless situation.

Fractures involving the air cells may allow, during straining, vomiting, etc., the escape of air into the superficial tissues, giving rise to pneumatocele or surgical emphysema. Occasionally air will enter the cranial cavity in sufficient volume to cause serious disturbance of brain function; such air pockets can be demonstrated by x-ray.

The hypophysis at times is involved in fractures through the sphenoid, and may give rise to a disturbance in its function such as occurs in polydipsia and polyuria.

SUMMARY

1. The base of the skull with its many foramina, their direction and the structures which pass through them, as well as the tightly adherent dura, must be carefully considered in fractures.

2. Fractures involving bones containing air cells may bring serious complications, particularly when this pneumatization is extreme. It may extend to the limits of any bone in any or all directions and the process may extend into adjoining bones.

3. The floor of the anterior fossa may consist entirely of pneumatized bone, the floor of the middle fossa almost entirely and the floor of the posterior fossa always consists partially of bone containing air cells. A basal fracture then must usually be considered compound unless proven to be otherwise.

4. The sphenoid is the "key-stone" of the skull, situated in the middle of the base and is in line of practically all basal fractures.

5. The temporal bone occupies a wide area in the base of the skull, and is unique in its connection with other bones. The arrangement is usuallly such that a certain amount of movement from below upward is possible.

6. Transverse fractures of the petrous pyramid usually extend through the labyrinth and seldom heal by bony union because the endochondral layer is not endowed with regenerative power. Thus in such cases there is constant danger of intracranial involvement in the event of upper respiratory infection.

7. The abducens nerve is placed in a very vulnerable position on account of its intimate relation with venous spaces throughout most of its long course, also on account of its relations with the basisphenoid and the petrous apex. It is very frequently involved in fractures through the middle fossa.

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XXV

TREATMENT OF TUBERCULOSIS OF THE TRACHEA AND BRONCHI*

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The question of tuberculosis of the trachea and bronchi is a very large one. This presentation is limited to one phase of the subject, the treatment of these lesions and their sequelæ. The subject is rather a new one and the best methods of treatment have not as yet been settled upon. In the course of the last three years, methods that were formerly used have been modified or even replaced by entirely different ones. Certain procedures have been found to be dangerous.

Tuberculosis attacks the bronchi in a number of different ways. In an active open case in which sputum contains numerous tubercle bacilli, shallow ulcerations may appear in the mucous membrane of the trachea or the main bronchi. These are thought to be caused by implantation. Such ulcerations are at times the cause of a persistently positive sputum, continuing to discharge tubercle bacilli even after the primary lesion has healed. At times the outer layers of a bronchus will be attacked by tuberculosis through the lymphatics. The disease spreads inward, perhaps involving the whole wall, attacking the cartilages and changing the bronchus to a mass of dense fibrous tissue with a small fistula running through it, representing the almost destroyed lumen.

Another form of attack is that through the mucous glands which lie in the outer layer of the wall of the bronchus. The disease spreads through the duct and appears in the interior of the bronchus as a spreading ulceration.

Closure of a bronchus can also be caused by pressure from the outside by a large tuberculous node. Such a gland at times will break down and ulcerate into the lumen of the bronchus. It will be understood that such an ulceration is a very different thing from an ulceration caused by implantation or spreading from a mucous gland. The final form which it may take is a widespread involve-

*Presented before the Eastern Section of the American Laryngological, Rhinological and Otological Society, Philadelphia, January 7, 1938.

ment of the mucous membrane. This later may become so thickened that its swelling almost closes the lumen of the bronchus. In some of these cases there will be no ulceration to be seen, only a thick congested mucous membrane lining the very narrow tube.

There is a great tendency for the body to heal tuberculosis. When this happens the tuberculous granulation tissue is replaced by dense scar tissue. Thus one of the sequelæ of tuberculosis of the bronchi is stricture from the contraction of this scar tissue, which may be so extreme as to completely close the main bronchus.

Let it be understood that the local treatment of tuberculosis of the bronchi is undertaken only with the co-operation of a well organized chest clinic. No bronchoscopist of himself should attempt to decide the many questions which will come up in the course of treating a case of tuberculosis, nor should he attempt any of the numerous surgical procedures which call for a highly trained specialist.

What had best be done in the way of local treatment? We know that these lesions will often heal spontaneously, therefore it may well be asked, should any local treatment at all be applied to them through the bronchoscope? It appears to the author that this question should be answered in the affirmative. As has been said, these ulcers when they heal produce scar tissue. It may successfully be maintained that those treated heal with less scar tissue and consequently less danger of stenosis than the untreated ones.

The bronchial walls are always in active motion. Therefore the first question I should like to raise is this: should not every case of ulceration of the bronchus have a pneumothorax done, in an endeavor to put the bronchial walls at rest, as is done in the treatment of tuberculosis in other parts of the body? As I understand it, a pneumothorax is done to close cavities in the parenchyma of the lung and in that way heal them. Is it not probable that the rest gained by a pneumothorax is as great a factor in the healing of a cavity as is the mere compression of it? At times there will be cases in which the parenchymal lesion will be so small as to be almost undetectable, yet the symptoms will be quite severe.

As an illustration of this point, let me quote the case of a woman of forty who was treated at Saranac for five months by rest in bed and feeding without favorable result on the bacillary content of the sputum.

The x-rays of her chest were negative, and yet she had considerable cough and a positive sputum. Bronchoscopic examination showed swelling of the mucous membrane and ulcerations and a

tuberculous tumor in the left main bronchus, just below the mouth of the upper lobe bronchus. She was treated bronchoscopically several times with applications of silver nitrate and of the mercury vapor lamp with very little effect on the extent of the ulcer, although the cough and sputum diminished. In spite of the fact that no cavity or other lesion was to be seen in the parenchyma of the lung, a pneumothorax was done with the idea of checking the respiratory movements of the bronchi. Only then did the ulcer begin to heal. (Figure 1.)

In contrast to this I might call your attention to the disastrous effect of leaving the lung expanded in the following case. In this case a young woman of 27 has had tuberculosis for the past five years. A pneumothorax was attempted but had to be discontinued four years ago. Then a left phrenectomy was done. About a year later the sputum became negative and has been negative since. However, she still has attacks of fever and periodic discharge of foul pus due to bronchiectatic cavities beyond a stricture. In spite of repeated bronchoscopies she continues to have these attacks. The chain of circumstances in this case, although of course one cannot be sure, has probably been as follows: It may be assumed that she had an extensive ulceration about the mouth of the left main bronchus. Since no medical aid was given, nature in the course of time healed the lesion, but with excessive scar tissue. The immobilization of the left lung by the pneumonectomy, since it prevented the air from entering the cavities cured the tuberculosis, for it has been demonstrated that tubercle bacilli deprived of oxygen will die. The cavities, however, became infected with anerobes, and not being sufficiently drained through the narrow fistula which had resulted from the contraction of the scar tissue, have persisted as bronchiectatic cavities. The thought is advanced that if the ulceration had been detected earlier by a bronchoscopy and a pneumonectomy carried out less severe scarring would have resulted.

Direct Treatment of Ulcerations.—The author in his earlier work treated these ulcerations by coagulation. This seemed to work very well indeed, healing taking place rapidly, yet much scar tissue formed. Then a disaster was met in the form of an uncontrollable hemorrhage, resulting in the death of a patient. If one could be sure that the ulceration had not approached the large blood vessels, coagulation would be perfectly safe. One could not be sure of that, so coagulation by diathermy has been given up.

Another method of treatment is the application of various chemicals. Several strengths of silver nitrate have been used up to 30%.

These seem to act very favorably on the ulcerations, bringing about a healing and reducing the swelling in the bronchus quite rapidly. Part of these effects are probably due to the fact that a good deal of secondary inflammation is present around the tuberculous ulceration and silver nitrate acts favorable on that. Silver nitrate seemed perfectly safe and has been used a great deal. The objection to silver nitrate is that it appears to produce an excessive amount of scar tissue which results in the closure of the affected bronchus. This may be a real disaster if the lung beyond the stricture is infected. Less irritating substance, such as chaulmoogra oil have been suggested. Some authors have reported very favorable results from this preparation in tuberculosis of the larynx. This would appear to be worth while trying, although Schugt, who had very extensive experience at the Sea View Hospital, Coney Island, N. Y., found it to have little effect.

The treatment which has appeared to this author to heal the ulceration most rapidly with the formation of less car tissue has been that with the mercury vapor lamp. Applications are made through the bronchoscope for two or three minutes every two weeks. Observation after a treatment shows a white fibrous film present over the lesion. Healing appears to take place quite rapidly. Treatment with this light is that most favored at the present time as it is safe and appears to heal the lesions with little scar formation.

Treatment of Contracted Bronchi.—As an example of this condition allow me to quote the following case. This woman had tuberculosis for a number of years. She had had a seemingly successful pneumothorax done, the lung appearing to be well compressed. Yet, there were still open cavities in the lung, and these contained a great deal of pus, free, however, from tubercle bacilli. The bronchoscopic examination showed the left main bronchus contracted down to a small fistula, blocked by granulation tissue. The treatment applied in this case was destruction of the granulation tissue, and suction of the lung beyond. At the first bronchoscopy a considerable amount of pus was removed with great relief of her symptoms. Repeated bronchoscopies were done, decreasing amounts of pus being removed at each one till finally the lung was completely dry. The question then came up, should an attempt be made to dilate the fistulous tract or should it be left to contract as it would. In spite of the bougies and glove stretcher dilators, it was found impossible to keep this stricture open more than to a certain extent. Finally, since the woman was symptom free, it was decided to do nothing more, and she has remained symptom free up to the present time. She is, of course, since the fistula is still open, always in danger of secondary infection with a

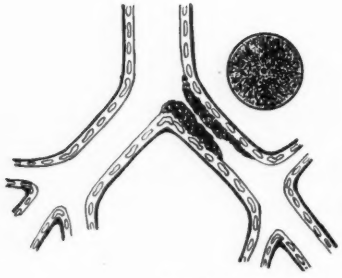

Fig. 1

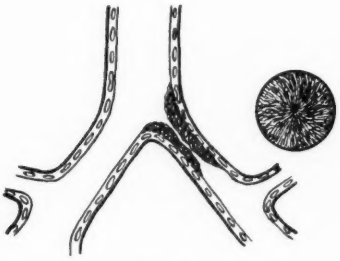

Fig. 2

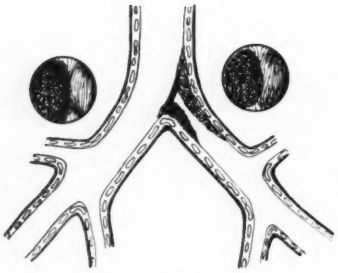

Fig. 3

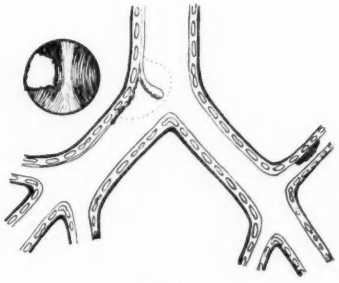

Fig. 4

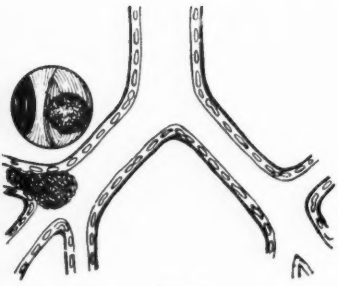

Fig. 5

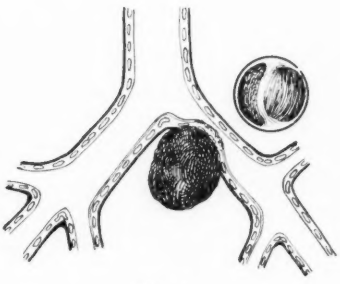

Fig. 9

renewal of the abscess formation in the diseased lung. The question thus arises, what is the best treatment now? Should she be left alone or should an attempt be made to close the fistula, either by applying strong silver nitrate to its walls or coagulating them. (Fig. 2.)

As an example of a complete closure of the bronchus, let me quote the following case. This woman had an extensive ulceration of the mouth of the left main bronchus. Her symptoms were quite severe, consisting of cough and the raising of sputum. The ulceration was treated with silver nitrate. It ultimately healed, the result being, rather unexpectedly, a complete closure of the left main bronchus. Through the bronchoscope it is almost impossible, at the present time, to demonstrate that a lumen ever existed on the left side, yet with the closing of the bronchus, the woman became symptom free and that is her present condition, no symptoms and absolutely closed left main bronchus.

Now the question arises, would not many cases in which the pneumothorax has been done and in which the lung is clear of infection benefit by the complete closure of the bronchus? If it so happens, either in cases with pneumothorax or in cases of thoracoplasty, that the lung has not been put completely at rest so that the air still enters and leaves the lung on respiration, distressing symptoms arise, consisting chiefly of a wheeze so loud as to be heard by bystanders in the room. Such a wheeze is very distressing to the patient. Would not one be justified in deliberately closing such a bronchus with the idea in mind of putting an end to the wheeze and protecting the lung against any further infection? Another effect may be expected from closure of a bronchus, that is the cure of tuberculosis in the cavities beyond. It has already been said that tubercle bacilli cannot live if deprived of air. Several authors have actually suggested the closure of bronchi as a means of healing tuberculous cavities. (Fig. 3.)

The procedure suggested in such cases is repeated bronchoscopies with aspiration until the lung is dry, then closure of the fistula by coagulating the walls. At times it is found quite difficult or impossible to render the lung dry. In such cases it may be necessary to dilate the strictured areas. This will be found very difficult to do on account of the toughness of the scar tissue. It is here suggested that ionization with the negative current be adopted. The author has been using a copper electrode with tips of different sizes to dilate these strictures. The electrode is inserted into the fistula and a negative current of three milliamperes is used for two or three minutes once a week. This has seemed to soften and dilate the scar

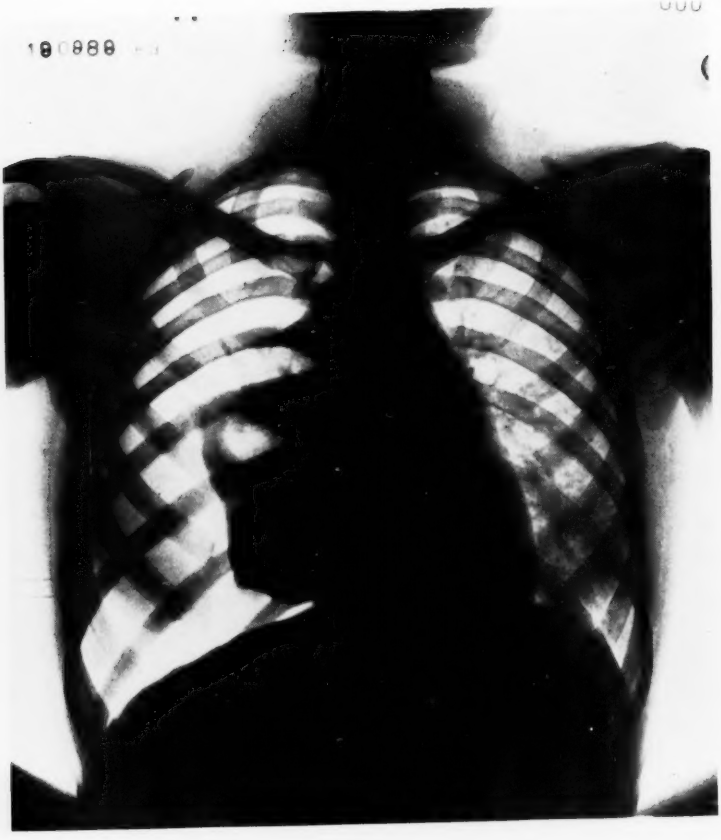

Fig. 6. Cavity before tumor was removed.

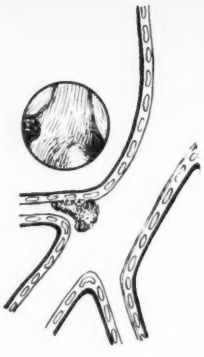

Fig. 7.

tissue. Following the dilatation, suction is carried out. It is proposed that if the lung is successfully dried, that the bronchus should then be closed. However, no fistula should ever be closed if there is active suppuration beyond it.

Tuberculous tumors as a rule attract attention through their tendency to cause blocking of some one bronchus. I have rarely seen one in the trachea or main bronchus large enough to close the lumen. Occasionally, however, when in these situations they will cause bizarre symptoms. In a case of a nurse who had been in poor health for several months, a state of affairs which had not been explained, the attention was drawn to her lungs by a peculiar fluttering noise heard at her mouth when she breathed. She was bronchoscoped and the fluttering was found to be caused by a pedunculated tumor, formed by the undermining of a strip of mucous membrane and its freeing from the surrounding tissue except for its attachment by one end. As it was considered dangerous to pull it away with grasping forceps, it was bitten away piecemeal. Only then was it realized that underneath it lay a ragged ulcer. Removal of the tumor of course relieved her of the fluttering noise and she was subsequently sent to a sanatorium. (Fig. 4.)

The most annoying tumors I have seen are those which form in the mouths of the secondary bronchi causing collapse of the lobe beyond. One patient, a woman, had what appeared to be a collapse of the right upper lobe. The bronchoscopy revealed a vascular tumor coming out of the mouth of the upper lobe bronchus. It was supposed that this was a carcinoma but section revealed it to be tuberculosis. Subsequent coagulations destroyed the visible part and a pneumothorax was successfully done. Occasionally the base of a tumor will be so far up a secondary bronchus that it cannot be entirely destroyed. Then, although the collapse of the lobe will be relieved, the annoying whistling during respiration may develop, much to the distress of the patient. The only recourse then is to an efficient pneumothorax, which stops respiration in that lung, or to a thoracoplasty. Occasionally the tumor will act as a check-valve, not entirely closing the bronchus, but trapping air so that it is retained in the lobe and helps in the formation of cavities or in their persistence even after pneumothorax. Such was the condition in a young woman who had had a pneumothorax done for tuberculosis in the right lung. (Fig. 5). In spite of the pneumothorax a cavity persisted. On bronchoscopy a small tumor could be seen in the mouth of one of the lower lobe bronchi; when this was destroyed sufficiently to allow free egress of air, the cavity immediately contracted. (Figures 6,

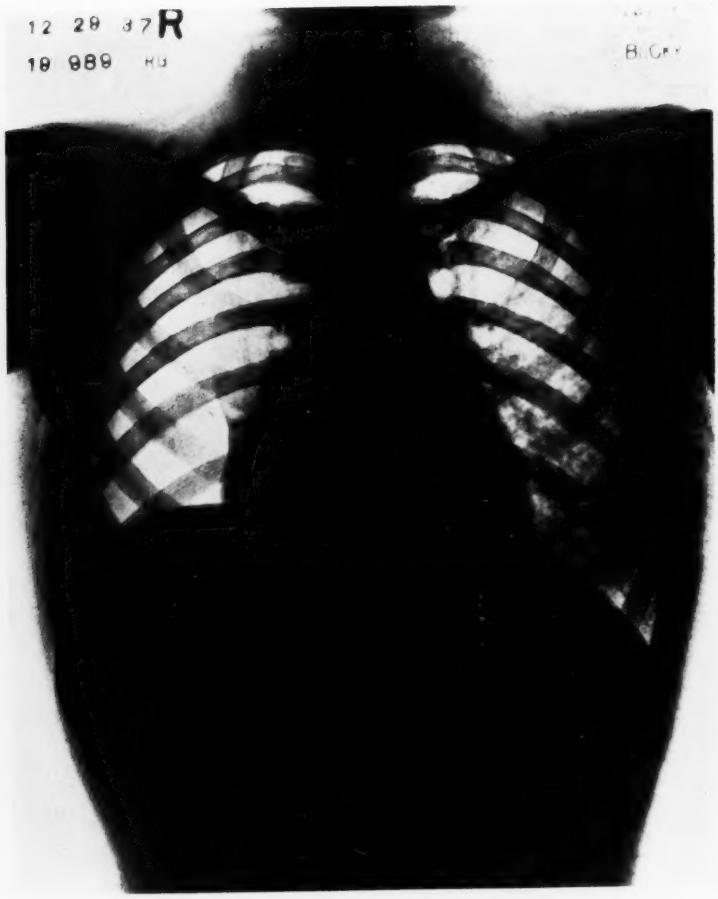

Fig. 8. Cavity after tumor was removed.

7 and 8.) The contention of some authors that where partial obstruction of a bronchus exists its total closure will aid in the collapse of cavities has already been mentioned, since air will no longer enter them to be trapped and help to blow them up. Also it has been said that tubercle bacilli deprived of air will die. Thus it is agreed that bronchoscopic closure of bronchi may be a valuable therapeutic measure. The case just quoted on the contrary demonstrates that there is no fixed rule about it. At times opening of a bronchus will aid in closing the cavity. One further question, what should be done in those cases in which, in spite of repeated aspirations and drainage a collapsed lung remains infected? Should we recommend a pneumonectomy or lobectomy? That is the question which can be answered only with the co-operation of the thoracic surgeon.

Treatment of Tuberculous Glands.—A tuberculous gland may contract the lumen of a bronchus and even at times close it. Probably the best treatment for these glands when they have not ulcerated in the lumen is deep x-ray therapy. (Fig. 9.)

Emergencies will at times arise in connection with tuberculous glands. An example is the rupture of a tuberculous lymph node into the lumen of a bronchus. This results at once in the collapse of the affected lung. The remedy in such a case is immediate bronchoscopy and clearing of the lumen of the bronchus by aspiration. In two instances the author has had occasion to remove from a main bronchus calcified lymph nodes which had ruptured through the wall and acted as foreign bodies. Once in the lumen they cause all the distressing symptoms of any other foreign body. A woman of 66 years of age had been treated for several months for asthma. She had a distressing wheeze and became so short of breath that cardiac decompensation was suspected. The appearance in the x-ray films suggested the possibility that a main bronchus might be compressed. The bronchoscopy revealed a calcified gland which had broken through into the lumen, so large as to almost completely block the right main bronchus and to encroach upon the trachea. This was successfully removed in several pieces with complete relief of her symptoms. Recently, however, she has had several attacks of bronchitis with wheezing and the x-ray films show a large gland which appears to encroach on the lumen of the right main bronchus.

The second case was that of a middle aged woman who had had wheezing for a year. There was a shadow in the right lung extending outward from the hilum to the base. The bronchoscopy showed a neoplastic mass in the main bronchus to the right lower lobe. Sections of this were reported as granulation tissue, though it was thought

to be tuberculous. The granulation tissue was coagulated and finally completely destroyed. It was then found that the base covered a calcified node which had eroded the wall of the bronchus but had not entered the lumen. An attempt was made to remove it but it could not be stirred from its bed. I should like to raise the question here, as to whether one should be justified in actually incising the wall of the bronchus and making an attempt to remove the gland at fault before it actually ruptures through.

To sum up, I have proposed in the first place, pneumothorax with immobolization of the lung when there is tuberculous ulceration of the bronchus. The ulceration is then treated, preferably by the application of the mercury vapor lamp. The chief sequela of the ulceration, provided it is successfully healed, will be contraction of the bronchus by scar tissue. Two methods of treatment are proposed in these cases in which the lung is successfully dried up. First, it is suggested that the bronchus be closed by coagulating its wall with the purpose of relieving any wheeze due to entrance and exit of air and to protect the lung from secondary infection. Secondly, these cases may be let alone. If it is necessary to maintain an open lumen during the course of aspiration, it is proposed to dilate the strictured bronchus by the application of a negative galvanic current, using the copper electrode. If it is found impossible to clear the lung of infection by repeated bronchoscopies the suggestion is made that such a lung might possibly be removed by pneumonectomy.

The treatment of tuberculous tumors, especially if they are obstructive, should be removal by coagulation. It is thought this procedure will be less likely to result in a spread of the disease.

Tuberculous glands may be reduced by x-ray. If they break down and form an abscess an incision through the wall of the bronchus is suggested. The author has no technique to propose for this procedure. Should they rupture spontaneously the pus of course must be immediately aspirated. Calcified nodes, if they rupture into a bronchus, must be removed as is any other foreign body. Incision of the wall of the bronchus for the removal of such nodes when obstructive, is tentatively suggested.

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XXVI

MIXED TUMORS OF THE HARD PALATE

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WATERVILLE, MAINE

The purpose of this paper is primarily to discuss the differential diagnosis of mixed tumors situated in the hard palate, and, in addition, to make certain observations suggesting their potential malignancy. While not especially common, the possibility of mixed tumor of the salivary gland type usually comes to mind when one encounters a tumor in this region. This is due, in part, at least, to the fact that a number of splendid papers on this subject have appeared in the literature in recent years. The histopathology and origin of these tumors have been studied by McFarland, Fay, Eggers, Ewing and others. Their conclusions have been summarized in two outstanding papers during the past few years. Sonnenschein's paper in 1930, while based upon mixed tumors in the soft palate, considered the broader aspect of the problem and included a very comprehensive review of the literature to date. In 1931, New and Childrey presented a most valuable paper on "Adenocarcinomas of the Mixed Tumor Type," based upon the study of 74 cases. Another valuable contribution to this general subject was made by Goldsmith and Ireland in 1936, although none of their six cases involved the hard palate. They devoted considerable attention to the matter of treatment.

There is more or less doubt as to the correctness of the term "mixed tumor" as commonly applied to these neoplasms of the salivary gland type, found occasionally upon the palate and less frequently elsewhere in the head and neck. New[1] classifies them as adenocarcinomas.

Sonnenschein[2] concluded that the exact origin of these tumors was not known, some authors believing that they are entirely epithelial and others that they are the result of accidental sequestration of embryonal cells. He felt that they had no relation to the normal structures in which they occur. New[1] says that the theory of embryonic displacement, or enclaves, is most commonly accepted. They are tumors in, but not of, the structures they invade. Goldsmith and Ireland[3], quoting Ewing, state definitely that they are not teratomas,

but that the extra-glandular type are probably from misplaced embryonal gland tissue. It is generally believed that the mucinous tissue and cartilage are derived by metaplasia from gland epithelium. In a personal communication Mallory[1] says, "Recent pathological opinion questions whether true cartilage is ever formed. I doubt if it can be proved that these palatal tumors are true mixed tumors, but they certainly resemble these more than anything else, and I think most laboratories call them mixed tumors." The general tendency is to consider them of ectodermal origin and due to enclavement of embryonal tissue.

New[1] says that these tumors present few diagnostic difficulties. They may occur at almost any age, but are commonest in the fourth decade. They usually have a history of being present for years without symptoms, except such as may be due to location and size. Usually these tumors are single, circumscribed, sessile and with a smooth nonulcerating suface. They may vary somewhat in consistency, depending upon the type of tissue contained. According to New, they may simulate inflammatory conditions, such as abscess; or gumma; or may resemble osteomata, or exostosis. They may be confused with sarcoma, or epithelioma, especially if there is involvement of the cervical nodes. Usually the diagnosis is suggested by the appearance and by palpation, but biopsy may be distinctly advisable.

Limiting our discussion to the hard palate there are other lesions which might well be considered in a differential diagnosis; especially with tumors of such small size as to make excision, rather than biopsy, the logical procedure. The complicated embryological development of this region lends itself to the formation of abnormalities. Up to the sixth week of fetal life the nasal pits and the primitive oral cavity are in open communication. With the descent of the frontonasal process and the inward growth of the maxillary process of the first visceral arch from each side, these three processes fuse, thus separating the nasal pits from the primitive oral cavity. Arrested development, or imperfect union may result in congenital defects; most manifest in the form of harelip, or cleft palate. This complicated development favors the enclavement theory—embryonic displacement—of the origin of the so-called mixed tumors. A partial defect in fusion might result in a pinching off of ectoderm from the primitive oral cavity and the formation of a cyst. A nasopalatine duct cyst may result from epithelial cell rests of a persistent nasopalatine duct. According to Goodman[5] these are more common than generally believed. Lipomata, fibromata or angioma are rarely, if ever, found in the hard palate. The fairly common osteoma is quite easily

diagnosed, but epithelial cysts, mucous cysts or adenoma may be confused pre-operatively with mixed tumors.

There is no unanimity of opinion in regard to the malignant characteristics of these tumors although generally they are considered as potentially malignant. It is agreed that there is a tendency to recurrence after removal, possibly years after. Eggers[6] considered malignancy of palatal tumors very rare, and felt that mixed tumors of the parotid only become malignant when they break through their capsule. Sonnenschein[2] considered the prognosis should be determined from the clinical history rather than the histology. New[1] goes definitely on record as considering all of these potentially malignant, as they may contain squamous cell epithiolma. The presence of enlarged cervical nodes would certainly suggest the possibility of malignant changes. Complete surgical removal, to be followed by post-operative radiation, if the histological examination shows malignant tendencies, is the generally accepted treatment. These tumors are not usually radio-sensitive and radiation alone is not generally satisfactory.

Four cases of similar clinical appearance but with different histology are reported.

REPORT OF CASES

CASE 1.—Miss J. B., age 36. Patient had noted swelling in the roof of her mouth for four years. This had not increased in size and was not painful or tender, although irritating from its size and position.

Examination showed a round, smooth, firm symmetrical tumor, about 1 cm. in diameter, at the posterior border of the hard palate, to left of midline. Examination was otherwise negative.

Operation: Novocain anesthesia, blocking the posterior palatine nerves. Mucosa incised and tumor shelled out by blunt dissection. Incision closed with one silk suture.

Macroscopic examination revealed, on sectioning, several small islands of what appeared to be cartilage, 1 mm. in diameter. Remainder of tumor was moist, glistening and fairly soft.

Microscopic examination (Dr. Tracy B. Mallory). "This specimen is characteristic of a fairly large group of tumors of the palate which, in their histology, resemble more or less closely the mixed tumors of the salivary glands. The epithelial cells occur in strands and cords, without definite gland formation, and are intimately admingled with a stroma, which is either hyaline or mucinous. It almost never, in my experience, looks as much like cartilage in these palatal tumors as it does in the salivary gland ones. This tumor is fairly highly cellular and shows an occasional mitotic figure, so that some degree of malignancy cannot be ruled out."

Diagnosis: "Atypical mixed tumor of salivary gland type."

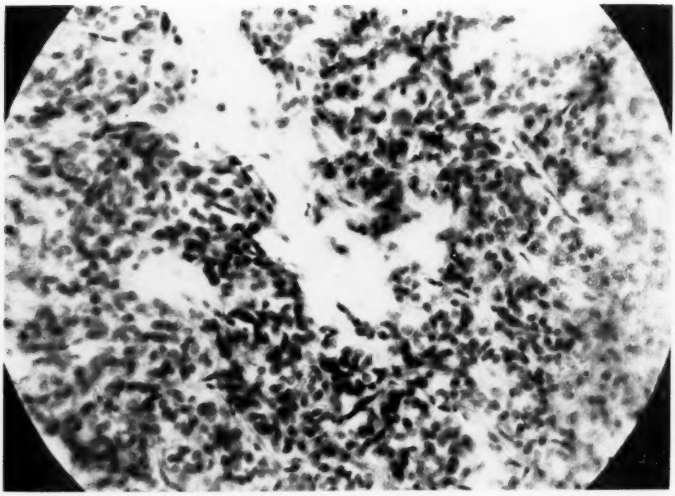

Fig. 1. Case 1. Mixed tumor, salivary gland type, palate. Occasionally mitotic figure seen.

Comment: While the histology of this case may be considered atypical, the clinical appearance and history is quite typical of this class of tumors, and bears out Sonnenschein's statement that the clinical history and appearance rather than histology should determine the prognosis. At the same time the microscopic findings corroborate the general opinion that these tumors are at least potentially malignant.

Case 2.—L. D., age 25. A swollen tender area had been noted in roof of mouth one week. This had increased in size. The patient consulted his dentist, who extracted several teeth in the belief that these might be the cause. Tumor was aspirated by family physician with negative findings, except for some brownish fluid.

Examination showed a tumor the size of a chestnut to the left of the midline of the hard palate just behind alveolar process. This was round, smooth and fairly firm.

Operation: Novocain anesthesia. Mucosa incised, tumor removed by blunt dissection. This exposed a round dehiscence in underlying bone, the tumor laying in this cavity in the palatal plate of the superior maxilla. The cavity did not communicate with the nose. Incision closed with silk sutures.

Macroscopic examination showed what was apparently a cyst with fibrous walls and containing brown colored serum.

Microscopic examination (Dr. H. E. Thompson). "Part of the specimen is composed of necrotic material with collections of blood cells and leucocytes, many of them polymorphonuclears. The rest is glandular tissue. There is no evidence of malignancy. The glands are composed of large pale mucous cells and appear to belong to the salivary group."

The patient made an uneventful convalescence and, according to reports from his physician, has had no recurrence in four years.

Comment: Obviously this condition was of longer duration than the history indicated. The clinical appearance suggested mixed tumor, although the location was quite consistent with a nasopalatine duct cyst. Operative findings would seem to indicate this latter as the diagnosis, yet this is disputed by the microscopic findings. There are many mucous glands in the hard palate but they are found posteriorly to the rugae, as a rule. The cyst must have originated in the boney cavity, remained quiescent for a long time and then suddenly began to expand downward. It might have been an inclusion cyst, resulting in the pinching off of aberrant glandular tissue during the process of fusion. It was probably secondarily infected, either from its location, or by the previous aspiration.

CASE 3.—Mrs. A. L., age 21. Three years previously the patient had severe pain in right side of palate. This area became swollen and finally broke down and discharged for a period of three weeks, after which it subsided. Swelling and pain had recurred five months before being seen. This was incised by her family physician. Pain and swelling had persisted since, with some discharge at times.

Examination showed a round, smooth, fairly firm tumor, about 1 cm. in diameter, on right side of hard palate, opposite first bicuspid tooth. A preoperative diagnosis of mixed tumor was made.

Operation: Avertin anesthesia, with novocain infiltration. Incision exposed what appeared to be a soft mass of granulation tissue, discharging pus. On removal a round opening, about 4 mm. in diameter, was found in the palatal plate of the maxilla. This opened into a cavity in the bone, about the size of a pea, lined with a membrane. This was cleaned out and the cavity packed with iodoform gauze.

Microscopic examination (Dr. Tracy B. Mallory): "The cyst is lined with columnar epithelium, but I cannot make out either cilia or mucous secretion. It might well have resulted from the dilatation of some small duct."

Diagnosis: "Epithelial-lined cyst."

Patient made an uneventful convalescence. The packing was discontinued after three days and the incision allowed to close.

Comment: A preoperative diagnosis of mixed tumor was made from the clinical appearance, although the history was suggestive of cyst. It seems doubtful if this had invaded the bone. It might possibly have been an inclusion cyst, resulting from some defect of fusion, although one would expect this to be in the midline. The microscopic findings were consistent with a nasopalatine duct cyst, and this would seem to be the most acceptable diagnosis except for its location. These are found anteriorly in the region of the papilla palatina where the anterior palatine foramen is situated. In this case there may have been an anomalous location, resulting in the more posterior situation of the cyst. This origin of the cyst is suggested by the predominance of pain as a symptom.

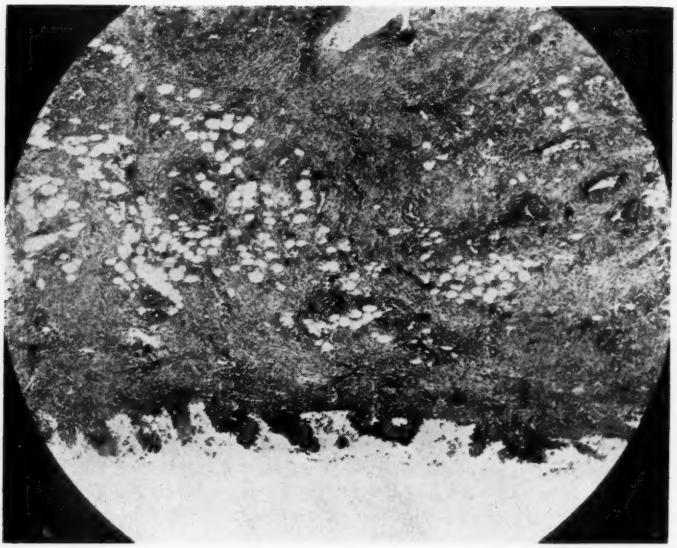

Fig. 2. Case 3. Epithelium lined cyst.

CASE 4.—S. B., age 55. Patient had had a tumor at the angle of left jaw for many years. This gave no symptoms until three months before being seen, when it began to enlarge. Shortly a complete facial paralysis developed.

Examination showed a firm nodular tumor in the region of the left parotid, extending below angle of jaw and pushing auricle somewhat upwards. This was not freely moveable. The overlaying skin was quite adherent. No secretion could be obtained from Stenson's duct. There was a complete facial paralysis.

Biopsy showed areas of carcinomatous changes in a mixed tumor.

The parotid gland was now removed under ether anesthesia. The lower portion was one indurated tumor mass which had penetrated the capsule and, in its center, containing apparently areas of cartilage. The upper portion showed some normal glandular tissue and contained some secretion.

Microscopic examination (Dr. Tracy B. Mallory): "The specimen shows obvious infiltrating carcinoma which is very poorly differentiated but distinctly more suggestive of an epidermoid than an adenocarcinoma. Paraffin sections show a large focus of hyaline and myxomatous tissue such as is commonly found in so-called mixed tumors. This seems to conform satisfactorily your clinical impression that the malignant tumor arose within an already existent, perhaps long standing, mixed tumor."

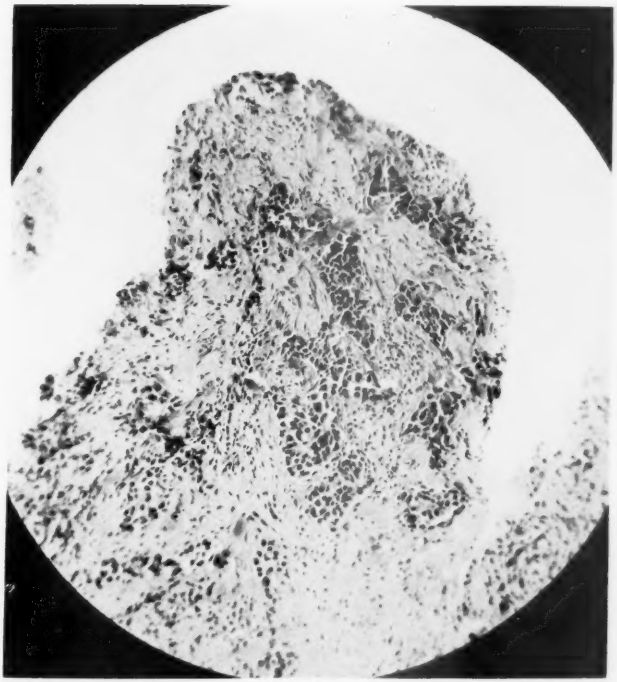

Fig. 3. Case 4. Carcinomatous involvement of mixed tumor of parotid. 110x.

Diagnosis: "Carcinoma."

Three months later the patient developed a Ludwig's angina and died, despite submaxillary drainage and tracheotomy. There was great difficulty in breathing, even after tracheotomy, and marked precordial pain. Postmortem examination showed a cervical cellulitis, acute mediastinitis and purulent pericarditis. An anaerobic streptococcus was obtained by culture. There was marked emphysema throughout neck and mediastinum.

Comment: While this case was not that of a palatal tumor, it is included in this series, as it shows carcinomatous changes in a mixed tumor of the salivary gland type. While this same tendency to malignancy may not be nearly so marked in the palatal tumors, the similar histological picture and the occasional findings of mitotic figures in the latter suggest at least a similar potentiality to malignancy. Possibly the difference in location may make a difference. The palatal tumors from their situation may expand without breaking through their capsules to an extent not possible in the parotid tumor.

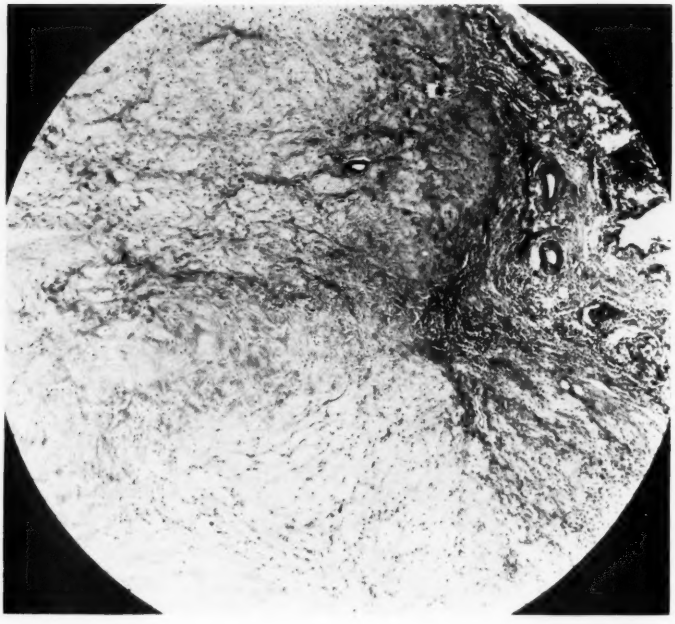

Fig. 4. Case 4. Carcinomatous involvement of mixed tumor of parotid. 50x.

CONCLUSIONS

1. While mixed tumors of the palate are usually diagnosed without difficulty, there may be confusion with tumors of small size in the hard palate when a biopsy is not done.

2. The embryological development of this region predisposes to the formation of abnormalities.

3. Epithelial or mucous cysts simulate mixed tumors in appearance. Diagnosis depends upon the microscopic findings.

4. Palatal mixed tumors are at least potentially malignant. While carcinomatous changes are far less common than in the parotid tumors the histology is similar and mitotic figures may be found.

5. The treatment is complete surgical removal—to be followed by postoperative radiation if there is any suspicion of malignant tendencies.

PROFESSIONAL BUILDING.

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XXVII

A FURTHER STUDY OF THE PATHOLOGY OF ACUTE LARYNGO-TRACHEO-BRONCHITIS IN CHILDREN*

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During the past ten years the clinical picture of acute laryngo-tracheo-bronchitis has been vividly painted by several experienced and competent observers. The seriousness of the condition, the importance of the unmistakable signs of upper respiratory obstruction and the urgent need of immediate relief have been so well emphasized as to require no further repetition in the present study. Despite this fact it is evident that the pediatrician and general practitioner still mistake the condition for diphtheria, spasmodic croup or some other milder form of laryngeal obstruction, with the result that these patients at times arrive at a hospital either as obstructive emergencies or in so toxic and exhausted a state as to render the hope of recovery a tragically forlorn one.

A consideration of the mortality rates reported by those men particularly interested in the condition and presumably thereby as likely as any to achieve success in its treatment is illuminating.

TABLE I

Name	Cases	Gross Mortality	Trache-otomies	Deaths	Tracheotomic Mortality	Date
Gittens[1]	32	39%	20	12	80%	1936
Smith[2]	43	9.3%	12	4	33%	1936
Baum[3]	24	41%	17	9	53%	1928
Author[4]	28†	40%	23	12	50%	1937
Total	127		72	37	51%	

In Table I are summarized mortality statistics from four separate series of cases of acute laryngo-tracheo-bronchitis. The lower gross mortality rate is obviously the result of inclusion of a certain number

*Presented before the Southern Section of the American Laryngological, Rhinological and Otological Society, Atlanta, Ga., January 24, 1938.

†I am indebted to Dr. Thomas Botsford for valuable aid in the compilation of statistics and to the Department of Pathology of the Children's Hospital, Boston, Mass., for assistance in preparation of the photomicrographs.

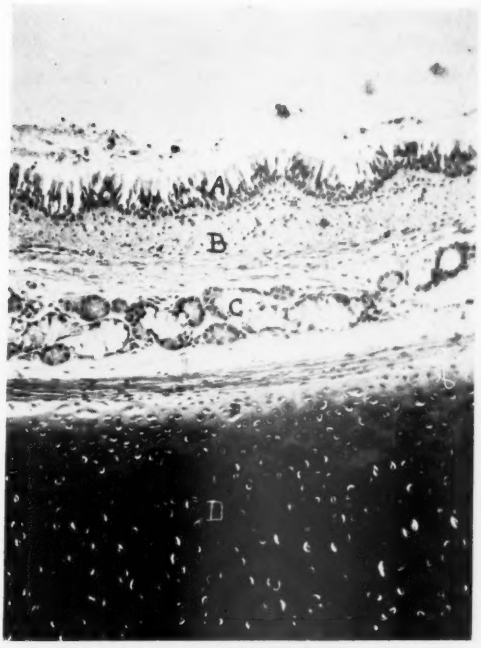

Fig. 1. Normal tracheal wall. *A*, ciliated columnar epithelium. *B*, tunica propria. *C*, mucous glands. *D*, cartilage.

of milder forms of infection which were relieved either without tracheotomy or by intubation. Considering the tracheotomized cases, the mortality averages 51%, a figure sufficiently high to warrant inquiry into any possible methods of treatment whereby it might be reduced.

Tracheotomy is still the procedure of choice among those most experienced with this condition with the exception of Baum.[3] In his series of 24 cases, seven were treated by intubation alone with only one fatality, while among his 17 tracheotomized cases there were nine deaths. Here, however, it is well nigh impossible to associate a mortality rate with a given operative procedure, since the severity of the infection may not have been comparable in the intubated and the tracheotomized patients.

In the most recent group of cases at the Children's Hospital in Boston, tracheotomy was performed in every case diagnosed as ful-

minating laryngo-tracheo-bronchitis. I feel that although intubation seems the easier and momentarily equally satisfactory procedure for the relief of the laryngeal obstruction, the risk of damage to the infected laryngeal tissues is so great as to make it inadvisable. Moreover, any further necessity for removal of lower tracheal and bronchial secretions necessitates tracheotomy if obstruction is to be relieved without serious laryngeal trauma, for which subsequent tedious dilatations may be necessary. There may well be good foundation for Baum's contention that the formation of sticky and gelatinous crusts is to some extent aggravated by air entering the tracheotomic wound. While this almost never occurs in hundreds of tracheotomized patients in the absence of acute infection, it may still play a part in laryngo-tracheo-bronchitis. Thus in one of the patients cited below (Case 3) who died before tracheotomy could be performed, all the elements of obstruction were present except crust formation. To date, however, considering the condition in all its aspects, tracheotomy remains an essential part of the treatment until some form of systemic medication or method of more rapid immunization can obviate the fundamental elements of edema and destruction of the mucosal surfaces. It is the desquamated epithelium from these latter that forms the nucleus for the obstructing masses which act like foreign bodies and still further diminish the size of the already restricted airway.

Among our most recent group of 17 patients with fulminating tracheo-bronchitis there occurred seven deaths. Of these, two may be excluded so far as operative procedure is concerned, since one (Case 3) was dead on arrival at the hospital and another died 42 days after tracheotomy from an influenzal meningitis. It is of great significance to note that in none of the remaining five fatal cases was a post-tracheotomic bronchoscopy performed. In the judgment of the various operators in charge, bronchoscopic aspiration was thought inadvisable or impractical in view of the patient's condition. Nevertheless, in three of the five, the postmortem examination revealed the definite presence either of "asphyxial membrane" or dry, tenacious masses of fibrinous secretion occluding the larger air passages. Autopsy was not permitted in the other two cases. Moreover, in two of these five cases there developed a pneumothorax, presumably the result of alveolar rupture in the presence of bronchial obstruction. While bronchoscopic removal of these obstructing masses might in no way have influenced the outcome in these cases nor had any beneficial effect on the concomitant mucosal edema, one cannot but feel that in any patient in whom clinical signs suggest obstruction of the airways, regardless of the seriousness of the clinical

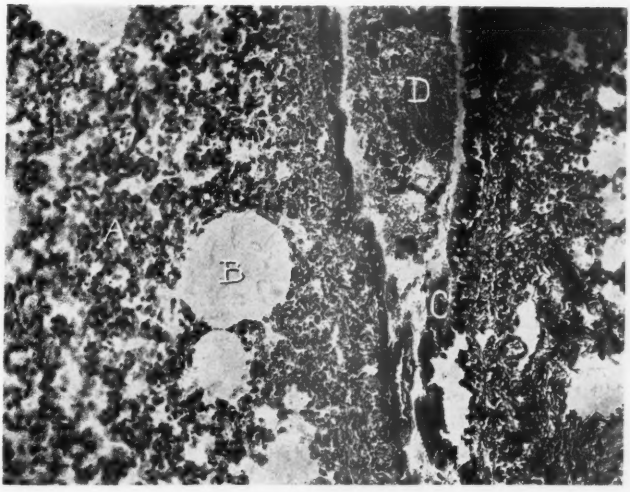

Fig. 2. Case 1. Lung and small bronchus (low power). *A*, area of atelectasis showing collapsed alveolar walls. *B*, area of emphysema with confluence of alveoli. *C*, disrupted bronchial mucosa lying free in the lumen. *D*, asphyxial membrane composed of amorphus eosinophilic hyalinized fibrin.

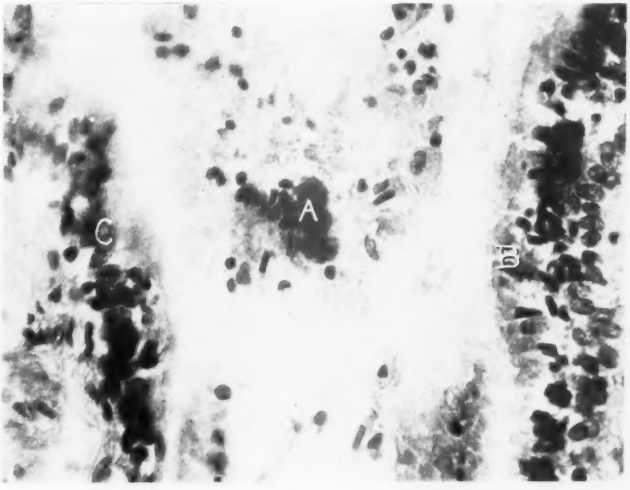

Fig. 3. Case 1. Small bronchus (high power). *A*, group of bronchial wall cells detached and lying free in the lumen. *B*, intact bronchial epithelium. *C*, disintegrating bronchial epithelium.

picture, at least one bronchoscopic examination is warranted as the only means of determining the possible existence of obstructing masses and of removing them, if present. To regard such patients as "too sick" to tolerate the procedure may at times be to forgo the one opportunity of saving the patient's life.

Obviously many cases will occur in which tracheotomy alone, with relief of the laryngeal obstruction, will be sufficient and bronchoscopy will be unnecessary. In the group of ten patients who recovered there was only one in whom subsequent bronchoscopy was required for removal of obstructing masses. This fact would seem to offer a strong argument against Baum's theory that it is the procedure of tracheotomy which is provocative of crust formation. The present low incidence of this complication may have been due to more zealous attempts to provide adequate moisture in the inspired air or to the use of solvents instilled directly into the trachea. It may on the other hand have been associated with a lowered virulence of the infection and a fortunate absence of secondary invasion by the staphylococcus organism.

In a commendably frank but discouraging reflection Gittens[1] in 1936 said: "After having recognized the nature of these non-diphtheritic infections for 16 years, and having tried every known method of treatment, our mortality rate is just as high as it was when a small number of cases were reported in 1926."

Inevitably one inquires, "Why is the mortality so high, and how can it be reduced?" Lucid descriptions of the clinical picture are available for any interested practitioner. Perhaps they need a wider dissemination, particularly among pediatricians and in pediatric literature. The cardinal therapeutic procedures are well recognized, their execution is a part of most well trained laryngologists' armamentarium, and well equipped hospitals everywhere have all the necessary facilities for operative and postoperative care.

Are we to look to the newer forms of systemic drug therapy for the hoped for reduction in mortality? In an infection in which the streptococcus hemolyticus plays such an important role it is but natural that sulfanilamide will be tried with increasing frequency. In the more recent cases at the Children's Hospital it has been used three times, with but one recovery. This in no way stamps it as a useless therapeutic adjunct, so varied are the factors entering into a case of severe respiratory infection. Further extensive trials of the drug are decidedly advisable with the hope that results may be achieved comparable to those in streptococcic meningitis.

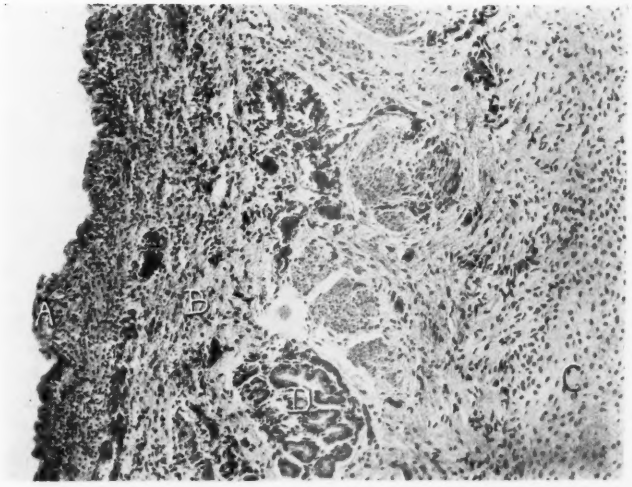

Fig. 4. Case 1. Trachea (low power). *A*, denuded tracheal epithelium covered with fibrin. *B*, edematous infiltration of tunica propria and submucosa. *C*, tracheal cartilage. *D*, mucous glands.

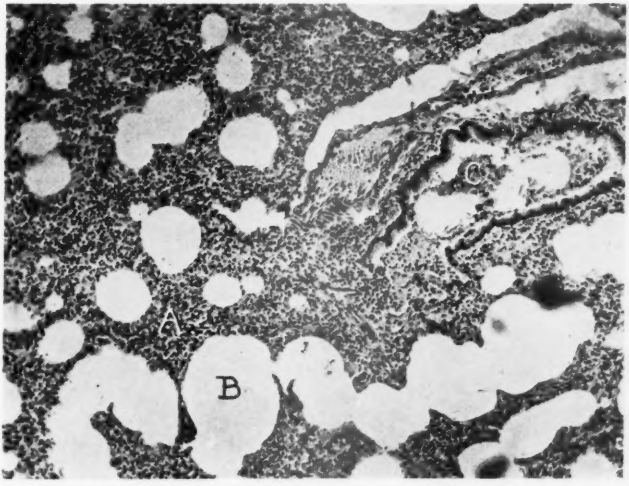

Fig. 5. Case 2. Lung (low power). *A*, area of atelectasis with collapsed alveolar walls. *B*, emphysema with confluent alveoli. *C*, small bronchus containing inflammatory exudate in the lumen.

In the meantime, there remains the less spectacular but always fundamental aid which may be derived from further study of the underlying pathological changes wrought by this disease. In 1933 I[1] presented some evidence to show the nature of these changes, particularly in respect to the destruction of epithelial surfaces of the trachea and the larger bronchi. It was felt at this time that the pathological changes in the lungs themselves were relatively slight. In the more recent series pathology in the alveolar structure has been more prominent and hence merits more careful study. One cannot escape the feeling that in many of the fatal cases in which no autopsy was permitted peripheral pulmonary changes may have played an important part in the incidence of the fatality. Terminal or even late x-ray films are not likely to be taken in these desperately ill patients and hence the pulmonary pathology escapes detection.

With the hope that a more careful study of postmortem findings may facilitate a clearer understanding of the underlying disease processes which work such havoc in creating a mortality of 50%, three cases are summarized and the essential postmortem findings illustrated by photomicrographs.

For purposes of comparison there is reproduced in Fig. 1 a view of the normal tracheal wall with its component parts. Attention is called to the clean cut regular ciliated columnar epithelium; the tunica propria, devoid of any evidence of edema or cellular infiltration; and the mucous glands, free from all signs of hyperactivity. It is these structures in part which so clearly manifest the changes incident to the type of infection under discussion.

REPORT OF CASES

CASE 1.—L. S., male, aged 1 year. November 10, 1935.

Complaint:

Allergic history. Thought to have asthma. Cyanosis and dyspnea. Temperature 102. Laryngoscopy—tracheotomy—transfusion. Convulsion—magnesium sulphate. Died, November 11, 1935. Cultures: Streptococcus viridans and staphylococcus.

Autopsy:

Gross.—Asphyxial membrane lining tracheal walls. Plugs of fibrin in bronchioles. Tracheal epithelium denuded. Cellular infiltration of tunica propria and submucosa. Mediastinal emphysema. Collapsed left lung. Atelectatic areas in both lungs. Edema of larynx.

Microscopic.—Lung (Fig. 2). Areas of atelectasis *A* combined with areas of emphysema *B*. Congested capillaries and infiltrating lymphocytes. Some alveoli contain red cells and phagocytes and walls are infiltrated with polymorphonuclear cells and lymphocytes.

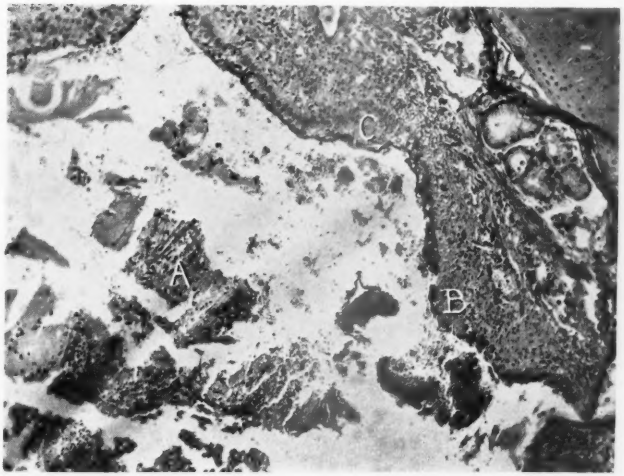

Fig. 6. Case 7. Bronchus (low power). *A*, masses of hyalinized desquamated tissue in bronchial lumen. *B*, Necrotic bronchial epithelium. *C*, bronchial mucosa with metaplasia to stratified squamous epithelium.

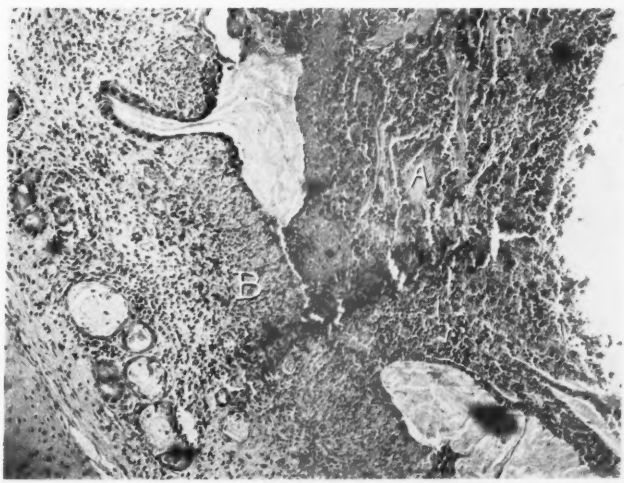

Fig. 7. Case 2. Lower trachea (low power). *A*, enormous masses of debris in tracheal lumen. *B*, tracheal mucosa with loss of surface epithelium and subepithelial edema.

Bronchus (Fig. 3). Mucosa disrupted *C* and free in the lumen. Asphyxial membrane lining alveolar and bronchial walls in form of amorphus eosinophilic hyalinized fibrin *D*.

Fig. 3. High power of bronchial wall seen in Fig. 1. At *A* is a group of bronchial wall cells disrupted and lying free in the lumen. On the right at *B* the bronchial wall is still intact, on the left at *C* it has lost almost all its normal continuity and is rapidly disintegrating.

Trachea (Fig. 4). At *A* the tracheal epithelium is denuded and covered with fibrin. There is seen at *B* a marked edematous infiltration of the tunica propria and submucosa, with scattered polymorphonuclear cells and lymphocytes. *C* cartilage. *D* glands.

Comments:

The classical picture of a fulminating case. Tracheotomy relieved only the laryngeal obstruction. Within the short space of 24 hours there has occurred, first a generalized edema and cellular infiltration of the trachea and larger bronchi, and secondarily a weakening and rupture of the alveolar walls, producing areas of emphysema, and finally with the advent of complete bronchiolitic obstruction, atelectasis and collapse of the alveolar spaces. No mere bronchoscopic plumbing and removal of desquamated epithelium and agglutinated fibrinous masses could hope to combat such a severe pathological reaction.

Case 2.—A. N., aged 15 months. March 23, 1937.

Complaint:

Cough, dyspnea, fever, six hours. Hoarseness. Treated by cough medicine. Marked distress. Croup tent.

Laryngoscopy—laryngeal edema. Culture, streptococcus hemolyticus. 3:30 P. M., tracheotomy. Still cyanotic. Suction—brown fluid and crusts. 6:15, much worse—cyanosis. Suction of no use. Sodium luminal. 7:30 P. M., left hyperresonance. ? pneumothorax. 10:00 P. M., oxygen tent. Sulfanilamide by mouth. X-ray shows left pneumothorax. Chest tapped repeatedly, q 4 h. Clysis of prontylin. Died 48 hours after admission. Blood culture positive for streptococcus hemolyticus.

Autopsy:

Gross.—Left pneumothorax. Left lung collapsed. Subcutaneous emphysema. Air in mediastinum. Trachea filled with thick tenacious mucoid material. At bifurcation is a thick paste-like mass of greenish yellow material obstructing both bronchi. Edema of bronchial mucosa. Right lung fills the pleural space. Emphysematous bleb in the mediastinum extending from the tracheal wound. Lungs and bronchi filled with thick tenacious yellow gray mucoid material containing many polymorphonuclear cells and G+ cocci in chains. Lower bronchi filled with same. Bronchial mucosa edematous and pink. Smaller bronchi show no purulent material. Larynx edematous—filled with thick, mucoid, tenacious yellow exudate.

Microscopic.—Lung (Fig. 5). Large lobular areas of atelectasis. *A* Alveoli are collapsed with smaller focal areas of atelectasis, and are filled with inflammatory exudate, polymorphonuclears and macrophages. Many alveoli are ruptured *B*, leaving scattered areas of emphysema between the atelectatic areas.

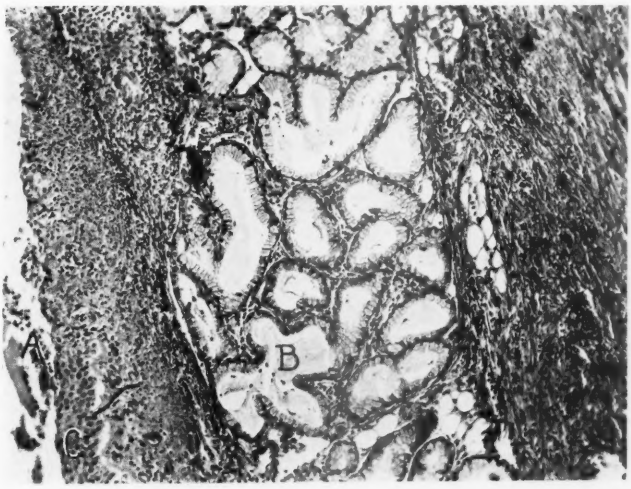

Fig. 8. Case 2. Upper trachea (low power). *A*, ulceration and necrosis of tracheal epithelium. *B*, dilated mucous glands. *C*, epithelial metaplasia to pseudostratified type.

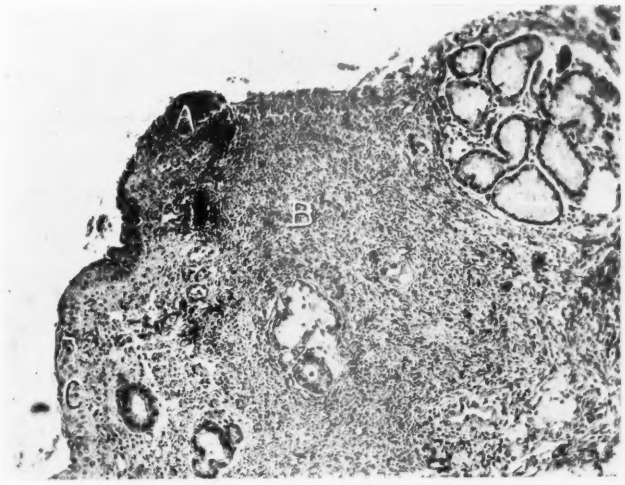

Fig. 9. Case 2. Larynx (low power). *A*, local ulceration and destruction of laryngeal epithelium. *B*, infiltration of submucosa with lymphocytes, plasma cells and polymorphonuclear cells. *C*, intact laryngeal epithelium.

Alveolar walls are inflamed and contain polymorphonuclear and plasma cells. Smaller bronchi *C* contain an inflammatory exudate composed of polymorphonuclears and macrophages. The bronchiolar epithelium is essentially intact, but there is some inflammatory cellular infiltration.

Bronchus (Fig. 6). The bronchial lumen contains large masses of hyalinized desquamated tissue *A* infiltrated with cellular products of inflammation. Focal areas of necrosis appear in the epithelial surface *B*, although portions are still intact. At *C* is seen the reaction already taken place of a metaplasia to a more stratified and squamous type of epithelium.

Lower trachea (Fig. 7). At *A* is shown an enormous mass of debris occupying the tracheal lumen. It is composed of desquamated epithelium and hyalinized fibrin, infiltrated with all the various cells characteristic of an acute inflammatory process. The tracheal mucosa is losing its integrity and the subepithelial tissue is intensely edematous, *B*.

Upper trachea (Fig. 8). Definite epithelial ulceration with separation of the necrotic surface cells *A*. The submucosal glands are dilated *B* and the walls congested with mucous. As in Fig. 6, there is a tendency to metaplasia of the epithelium to the pseudostratified type *C*.

Larynx (Fig. 9). A section through the wall shows an area *A* of local ulceration and destruction of the epithelium. The underlying tissue *B* is markedly infiltrated with lymphocytes, plasma cells, macrophages and polymorphonuclears. An area of intact stratified laryngeal epithelium is seen at *C*.

Comments:

This patient was not bronchoscoped subsequent to the tracheotomy. In view of the pathological findings with large obstructive masses in the main bronchial tree, this seems somewhat surprising. The patient, however, was so desperately ill and the pneumothorax was so prominent a symptom that attempts to deal with this aspect of the case overshadowed all other considerations. Nevertheless in retrospect it would seem that regardless of the severity of the illness every attempt should be made to maintain the patency of the upper airways, otherwise the inevitable atelectasis is likely to prove fatal. This case showed the additional finding of a true unilateral pneumothorax, a condition which merits some special consideration, both from an etiological and pathological standpoint. The pathological report states that no evident break in the pleura was discovered and yet the mechanism of non-traumatic pneumothorax depends on such a rupture, however small, through which air may enter the pleural cavity.

Smith[2], in his report of 43 cases, mentions three fatalities, two with unilateral and one with bilateral "pneumothorax." However, he also cites two cases of "pneumothorax" reported by Tucker[5] in an article on "Pulmonary Atelectasis," whereas reference to the original paper clearly stamps the condition as atelectasis with no mention whatsoever of pneumothorax. This confusion of terms naturally leads one to inquire whether in Smith's own cases four instances of "pneumothorax" should in reality be construed as obstructive atelectasis.

Simpson[6] reports a case of pneumothorax complicating tracheotomy in fulminating tracheobronchitis, the first of its kind (Smith's report notwithstanding) which he was able to find. Numerous instances of pneumothorax as a postoperative complication of tracheotomy are on record, the theory being that if the pre-

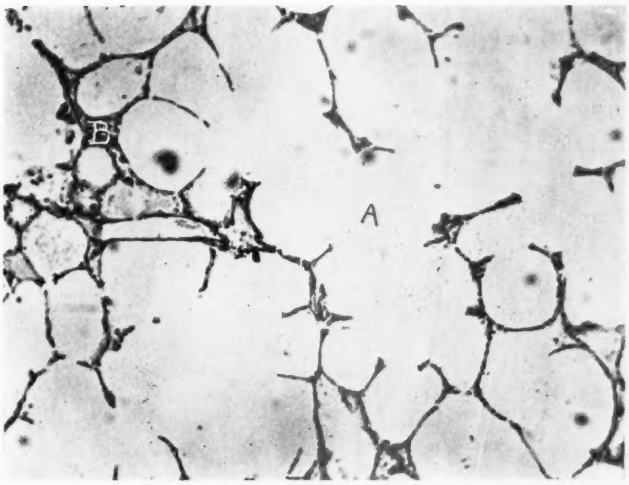

Fig. 10. Case 3. Lung (low power). *A*, enormous confluent alveolar areas producing emphysema. *B*, inflamed and infiltrated alveolar walls.

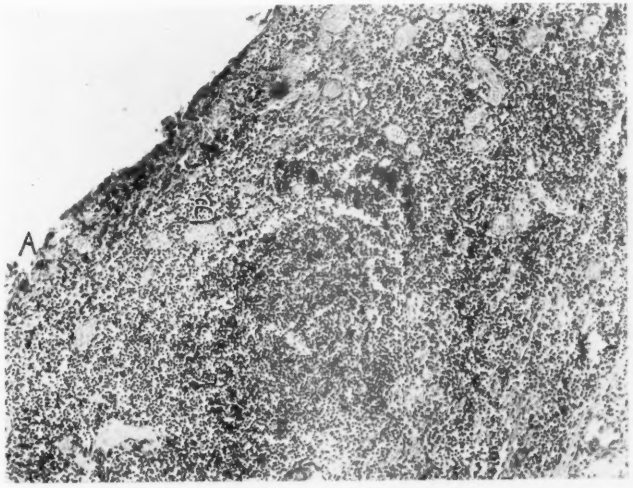

Fig. 11. Case 3. Bronchus (low power). *A*, denuded bronchial epithelium. *B*, tunica propria infiltrated with polymorphonuclear and lymphocytic cells.

trachial fascia is too extensively separated, air may enter the mediastinum and, if under sufficient pressure, rupture the pleural surface and so enter the chest cavity.

In Simpson's case, right sided pneumothorax developed 24 hours after a tracheotomy was performed, under ideal conditions, for laryngo-tracheo-bronchitis. Repeated right bronchial obstruction by crusts necessitated some 50 bronchoscopies with ultimate recovery. Simpson does not explain the etiology of the penumothorax, but it seems quite logical to conclude that with such marked bronchial obstruction an emphysematous dilatation of a peripheral alveolus could cause rupture into the pleural cavity without in any way incriminating the procedure of tracheotomy. Surely the same mechanism is seen from time to time in whooping cough with pleural rupture due to violent coughing.

Pneumothorax was found in Case 2 and also in the following case, indicating that this complication of laryngo-tracheitis is not as rare as Simpson's report would lead one to believe. In our cases the diagnosis was definitely established by antemortem aspiration.

R. B., aged 2 months. Two previous acute upper respiratory infections within a year. Admitted with croupy cough. Temperature 101 degrees. Dyspnea marked. Tracheotomy. Condition progressively worse. Right pneumothorax; repeated aspiration of air. Clysis of sulfanilamide. Died. No autopsy.

It is obvious that the complication of air in the pleural cavity with resultant compression of the lung adds a further burden to an already seriously embarrassed respiratory function. Moreover, it is a condition which is not amenable to any satisfactory therapy. Tapping and repeated aspiration are but temporary in their effect so long as air continues to be forced into the pleural cavity with each expansion of the chest. To a swollen bronchial membrane and alveolar atelectasis from the resultant obstruction, a pneumothorax adds an almost insurmountable complication.

Case 3.—C. M., aged 5 years. January 12, 1934.

Complaint:

Anorexia, head cold, fever, 24 hours. Severe dyspnea. Dead on admission to hospital. No operation.

Autopsy:

Gross.—Lungs, crepitant. Trachea and bronchi contained thin yellow exudate but no obstructive crusts. Severe cellulitis of the whole upper respiratory tract. Inflammatory involvement of terminal bronchioles.

Microscopic.—Lung (Fig. 10). A high degree of emphysema, with confluence of the alveolar walls converting the normally segregated spaces into great lakes resembling a countryside flood. In places the alveolar walls are inflamed and infiltrated with cells of various types *B*. This breakdown of alveolar partitions can only be the result of obstruction in the smaller bronchi, preventing the egress of air and aggravated by the patient's effort to carry on respiratory efforts. In spite of this weakness of the alveolar walls, there was no pneumothorax in the case.

Bronchus (low power). (Fig. 11). The epithelium of the bronchial wall is denuded *A*. The tunica propria is extensively infiltrated with polymorphonuclear and particularly lymphocytic cells *B*. The picture so far as the tissues go is the

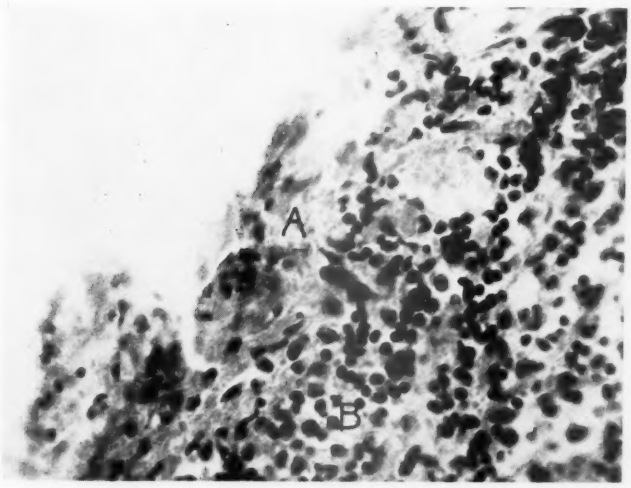

Fig. 12. Case 3. Bronchus seen in Fig. 10 (high power). *A*, denuded epithelial surface, edema and inflammatory cells. *B*, submucosa infiltrated with numerous lymphocytes.

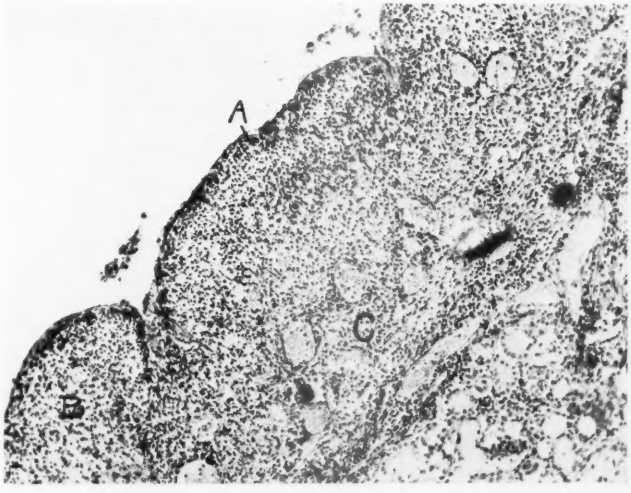

Fig. 13. Case 3. Trachea (low power). *A*, denuded tracheal epithelium. *B*, edematous tunica propria. *C*, submucosal infiltration with lymphocytes and polymorphonuclear cells.

same as that seen in Cases 1 and 2, with the absence of definite cellular debris in the lumen. Fig. 12 (high power) shows clearly the change in the surface epithelium. All normal columnar epithelium *A* is lost, replaced by products of a severe cellulitis with edema and infiltration by reactionary cells *B*.

Trachea (low power). (Fig. 13). Denudation of the normal tracheal epithelium *A*, edema of the tunica propria *B*, and infiltration of the submucosa with lymphocytes and polymorphonuclear cells *C* extending deep down toward the submucosal glands.

Comments:

This case is of particular interest since it is the only one in our recent series in which no operative measures for relief of obstruction were carried out. This patient, seen several times by the local physician, was finally rushed to the hospital as a desperate emergency and died before arrival. The question of immediate tracheotomy performed perhaps even at the roadside is of course easily raised, but in view of the pathological findings, it is highly improbable that it would have been of more than transitory value. The pathologist stressed in this case particularly the complete absence of crusts or accumulations within the tracheal or bronchial lumen and the high degree of obstruction resulting solely from the constricting edema of the walls of the larger and smaller airways. This finding of course lends some support to the theory first advanved by Baum that tracheotomy, by permitting the direct exposure of the tracheal exudate to the air, is instrumental in the formation of the tenacious and obstructing crusts. Whenever some method is devised of reducing the inflammatory edema of the respiratory mucous membrane it may be that tracheotomy can be dispensed with. Certain it is that something must be done to relieve the laryngeal obstruction and at the same time to remove even fluid tracheal and bronchial secretions in order to permit adequate oxygenation of the lung. Perhaps helium will prove to be the answer supplemented by chemotherapy. At present any measures which will counteract dryness in the inspired air must be employed, together with solvents of obstructing masses when they form. As yet we have found nothing quite so effective in this respect as a solution of sodium perborate, frequently instilled into the tracheotomic tube and removed soon after by catheter suction. In this way the main airway can often be kept patent pending other measures designed to combat the infection.

CONCLUSIONS

1. Acute fulminating laryngo-tracheo-bronchitis remains a serious respiratory infection in children, often erroneously diagnosed, the urgent need of its relief not appreciated and its mortality distressingly high.

2. Pneumothorax, secondary to pulmonary emphysema, is a complication present more frequently than hitherto appreciated.

3. Tracheotomy is still the ideal method of relief of laryngeal obstruction, but the lower airways must if necessary be kept free from obstructive material by repeated bronchoscopies.

4. Further trial of sulfanilamide is needed to establish its worth in this infection.

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XXVIII

HISTOPATHOLOGY OF THE PERIPHERAL AUDITORY MECHANISM IN DRUG-INJECTED ANIMALS*

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The results of several years of study on the effects of drugs on the peripheral auditory mechanism will be summarized and presented in this paper.

The investigations have been carried out at the George Williams Hooper Foundation and under the Coleman Memorial Fund of the University of California. The observations to be reported have been published previously or are in press at the present time.[3, 4, 5, 6]

The drugs selected for study were those the use of which had been known to result in deafness and instances of their effects have appeared in the literature. They included quinin, salicylate, barbiturates, codein, strychnin and others. In Taylor's[10] recent review of the literature on drugs and chemical poisons causing nerve deafness, tobacco, alcohol, arsenic, carbon disulphide, oil of chenopodium, mercury, morphin and anilin dyes are also included. It is obvious that the nature of these agents is varied and presumably their actions upon various parts of the peripheral auditory mechanism are also different. It becomes the problem of the investigator to determine the location and nature of the lesions produced by these agents and when possible to correlate these with functional hearing tests.

Any number of demonstrable lesions due to the action of certain drugs could incapacitate the auditory mechanism. The structures contained within the membranous cochlea and particularly the hair or sensory cells of the organ of Corti are extremely susceptible to the actions of various agents. Damaged hair cells theoretically imply faulty reception of stimuli and transmission of impulses in the audit-

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*From the George Williams Hooper Foundation, University of California, San Francisco, California.

Supported by a grant from the John C. and Edward Coleman Memorial Fund, University of California.

Presented before the Western Section of the American Laryngological, Rhinological and Otological Society, Santa Barbara, January 30, 1938.

ory nerve. The complete story of the manner in which various agents impair hearing does not lie in a study of the hair cells alone. Many other contributing factors such as evidence of altered cochlear fluid pressures are of significance. The review will be confined to the histopathological changes in the peripheral auditory mechanism induced by quinin and salicylate because these drugs are commonly used and because of the fact that deafness frequently results from their administration.

HISTOPATHOLOGICAL STUDIES

The primary effects of quinin and salicylates have been attributed to the ischemia resulting from a constriction of the blood vessels, either peripherally, centrally or both, in the auditory mechanism. Quinin has also been termed a "direct protoplasmic poison." While ischemia may be present in the early stages of an intoxication due to either of these drugs, constriction of the vessels within the stria vascularis is not present for animals which have received drugs over a period of several weeks or months. To ascertain in quantitative terms the amount of distention of the vessels in the stria vascularis estimations of the relative vascular areas in terms of the total surface area were made. It was found that for control animals approximately 6% of the stria vascularis surface area was presented by blood vessels while in drug-injected animals this estimate was more than doubled (12.5—13.0%). This observation taken in conjunction with the hypothesis of Belemer[1] on the control of intracochlear fluid pressures by the spiral ligament and stria vascularis may give evidence that the pressures within the cochlear duct are altered by these drugs.

The origin of the fluid has usually been attributed to the stria vascularis. Shambaugh[8] believes the epithelium of the external spiral sulcus cells and the neighboring blood vessels comprise a glandular organ responsible for the endolymph. It may be found that the stria vascularis represents an absorbing surface in the regulation of the fluid pressure while the fluid itself has its origin in the region of the external spiral sulcus. The mechanism would be somewhat analagous to the function of glomerular tufts of the kidney with the stria vascularis comparable to the convoluted tubules. Curiously the cytological constituents and appearance of the cells within the stria bear some resemblance to those within the convoluted tubules of the kidney.

The mitochondria were found to serve as an index for the action of quinin or salicylate upon the cells of the stria vascularis. Preg-

nant guinea pigs which received quinin or salicylate during the period of gestation revealed degenerative changes in the mitochondria of the cells of the stria vascularis, while in the fetal guinea pigs there were even more pronounced changes in these cells.

The external sulcus cells in these drug-injected animals were found to be swollen and vacuolated although there was no evidence that mitochondria had been damaged.

Degenerative changes in the external hair cells were commonly encountered in animals which received either quinin or salicylate. The internal hair cells were apparently more resistant, a factor which lends support to the contentions of Lurie[7] that functionally these two types of vibroceptors are different. Cytologically, at least, the external hair cells represent a more specialized type of cell. For this reason it might be expected that they would first reveal degenerative changes of the type to be described. The external hair cells become vacuolated, distorted in shape, and the fine rod-like mitochondria of the normal cell become droplets. Similarly the endings of the cochlear nerve when studied by supravital methylene blue staining also reveal alterations. At first the terminations become swollen and stain faintly with the dye, but in more advanced stages their continuity is lost and they appear as a blue granular substance in the base of the cell.

The myelin sheath of the cochlear nerve fibers has been shown to be exceptionally unstable and degenerative changes in it are produced by a variety of factors. Even so-called normal or control animals exhibit varying degrees of changes in the myelin. The nerves of animals which received a series of injections of quinin or salicylate were found to exhibit degenerative changes in the myelin sheath more extensively than those encountered in control animals of the same species and litter. After 57 daily injections of quinin the myelin sheath became markedly altered while after 76 daily injections an abundance of blackened segments of the myelin sheath was produced. These changes extended from the periphery of the nerve, gradually became pronounced in the auditory nerve proper.

Changes in the spiral ganglion cells due to drugs, toxins or poisons have probably been more often described than for any other part of the neural mechanism. Usually the nature of the alterations is found to be vacuolization of the cytoplasm, loss of the chromatophilic substance and tendency to displacement of the nucleus. Alterations produced by quinin and salicylate were of this nature and more severe for the latter drug.

COMMENTS

From this brief review of the histopathologic changes resulting in the peripheral auditory mechanism of animals injected with quinin or salicylate it is obvious that the action of these drugs is not only confined to the vascular supply to the cochlea, but is apparent throughout its entire structure. Any one of the changes described might be sufficient to damage or impair hearing to some degree although these findings in animals remain to be correlated with electrical hearing tests.

It has not been shown that the central auditory pathways and the auditory area in the cerebral cortex are affected, but certainly the presence and extent of the lesions produced in the peripheral auditory mechanism should justify caution in the use of these drugs.

Taylor[10] has stressed the danger of the use of quinin or salicylate in pregnant women. Both drugs have been shown to pass readily into the fetal circulation, and hence the hearing of the child may be impaired as well as that of the mother. In addition, quinin has been associated with death of the fetus. Taylor believes that quinin should be administered with the utmost caution to persons who have a hereditary tendency to deafness. Under such conditions its use should be contraindicated in pregnant women.

On the other hand Watson[11] in discussing Taylor's paper at the American Otological Society meetings in May of last year stated: "In no case that has come under my observation has there been any complaint of a permanent ill effect on the auditory apparatus of the mother." He concluded,—"there is not sufficient clinical evidence that this (quinin) may cause either the death of the child or permanent injury to the auditory apparatus to warrant discontinuing the use of this valuable drug in practice."

While the clinical evidence for the relative importance of these drugs as agents in the production of deafness lacks a comprehensive survey, the conservative stand of Taylor seems justified. It is the pregnant woman with a family history of deafness for whom the use of quinin should be contraindicated as well as the occasional individual with idiosyncrasy for quinin or salicylate and the already deafened individual.

Not infrequently the residual hearing of an individual becomes noticeably decreased by the administration of a drug for one purpose or another. Coleman[2] has made a plea that the clinician become, with respect to individuals who already suffer a hearing loss, more deafness-minded in the use of drugs.

Shurly[9] has recently emphasized the importance of otolaryngology in relation to general medicine. He believes that otolaryngology should lend "a helping hand" in the detection of the early manifestations of certain diseases such as organic heart disease, tuberculosis, cancer, pneumonia and diabetes. The reverse situation is also of importance with respect to the use of certain drugs by the clinician who usually prescribes them without consideration of their possible effects upon the auditory mechanism and especially upon that of a deafened individual.

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XXIX

AN ANALYSIS OF 75 CASES OF BRONCHIECTASIS FROM THE VIEWPOINT OF SINUS INFECTION*

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The purpose of this paper is to present the clinical findings with special reference to the role of sinus infection obtained from a study of 75 cases of bronchiectasis at the Pulmonary Clinic of the Massachusetts General Hospital. Three years ago it was decided to assign a laryngologist to this clinic in order to carry on investigation regarding the etiological role of sinus infection, and to arrive at a more definite understanding of the importance of sinus infection when present in cases of bronchiectasis. Through the kindness of Dr. Mosher this assignment was offered to me and here at the beginning of this paper I wish to thank him for his help throughout this investigation.

Before the cases are referred for inspection of the nose and throat they have been given a complete general examination, including an x-ray of the chest after injection with lipiodol. I have not included in this study any cases where there is any doubt in regard to the presence of bronchiectasis. The nose and throat investigation consists of the routine procedure followed in this hospital. It includes a careful history and an examination of the nasal passages, postnasal space, pharynx and larynx, and transillumination and x-ray of the sinuses. Lipiodol is not used in the x-ray of the sinuses, as it has been found that this procedure in our clinic adds little in the way of diagnosis to what can be discovered by Dr. MacMillan's usual x-ray technique. In approaching this problem, the following questions presented themselves:

1st. To what disease does the patient attribute the onset of his pulmonary infection?

2nd. Do the cases show any tendency to arrange themselves in corresponding etiological groups?

3rd. What is the incidence of sinusitis for each such etiological group?

*Presented before the 60th Annual Meeting of the American Laryngological Association, Atlantic City, May 2, 1938.

4th. Does the incidence of sinusitis in each such etiological group vary with the ascribed etiology?

5th. Does the sinus infection bear any relationship to the extent of the bronchiectasis?

6th. Whether or not any sinus infection was present at the onset, does it play any part in the subsequent course of the pulmonary disease?

To simplify the data in a study which is essentially statistical, I am going to present it in four sections. The first section is intended to give the general picture of this series and is mainly a matter of vital statistics. The second section gives the etiology as obtained in the histories and reasons for considering the several diseases as distinct etiological groups. The third section correlates the incidence of sinus disease with the etiology and the fourth section correlates the sinusitis with the severity and extent of the bronchiectasis.

In the first two charts I will show you the material on which the study is based.

CHART I

Number of Cases—75

Male	34	45⅓%
Female	41	54⅔%

Age by Decade at Onset of Bronchiectasis

Decade	1st	2nd	3rd	4th	5th	6th
No. of cases	24	20	10	7	5	3

The incidence of bronchiectasis is slightly higher for women. It is interesting to note also that bronchiectasis is much more likely to occur in the first two decades of life.

CHART II.

DURATION OF THE BRONCHIECTASIS AT THE TIME OF EXAMINATION

1 year or less	5 cases
2 years	7 cases
3 years	8 cases
4 years	4 cases
5 to 10 years	22 cases
11 to 20 years	16 cases
21 to 30 years	6 cases
7 cases undetermined	

In this series 65 per cent of the cases at the time of examination have had their bronchiectasis for more than 5 years. Only 5 cases have had it for a year or less.

By the time these patients come to us for a nose and throat examination, the bronchiectasis is frequently of several years' duration, and sinusitis, if present at all at the onset of the pulmonary disease, therefore has had plenty of time to become established as a chronic infection. The greater number of cases, 65%, have had their pulmonary disease for more than five years as is shown in Chart II. With these few vital statistics in mind, I shall now discuss the diseases to which the patients attribute their bronchiectasis.

CHART III.

Disease at Onset		No. of Cases
Upper respiratory infections	Head colds	21
	Sinusitis	6
	Head colds with pneumonia	5
Total		32
Pneumonia		21
Influenza		5
Asthma		2
Bronchitis		1
Whooping Cough		5
Tb. Contact		1
T. and A.		2
Unknown		6
Total		75

The classifications of upper respiratory infection, "sinusitis" and "colds with pneumonia" can be grouped together and account for 42.6 per cent of this series. Pneumonia alone is the next most frequent etiological disease.

By "upper respiratory infection" is indicated the group of patients that give "head colds" but not "sinusitis" in the history. It may be reasoned that the classification of upper respiratory infections as distinct from sinusitis is an unimportant one here. However, it does indicate degrees of severity. I have put it down here in a separate group intentionally to emphasize the fact that only six of these cases could state positively that their bronchiectasis followed a sinus infection.

There is also another classification which should be explained, that of "colds with pneumonia." I realize that some observers believe that all cases of pneumonia have an onset preceded by an upper respiratory infection. But these 21 cases of "pneumonia" could not recall any preceding upper respiratory infection. Only five cases were able to remember a head cold occurring at the time of their pneumonia.

I am inclined to place these "colds with pneumonia" cases with the sinusitis group, as without exception they all have a chronic sinusitis. Also, in order to have a clear-cut differentiation of etiology I wish to keep the 21 cases of "pneumonia alone" as a distinct group.

So far, I have considered the disease occurring at the onset of the bronchiectasis, the age at which the bronchiectasis began, and the relative proportion of the two sexes. On checking these three factors against each other, I found some rather striking relationships.

CHART IV.

Disease at Onset	Age at Onset by Decades (No. of Cases)						
	1st	2nd	3rd	4th	5th	6th	? Decades
Upper respiratory infections							
Head Colds	2	8	4	2	2	2	2
Sinusitis	2	3		1			
Colds and Pneumonia	1	1	1	2			
Total	5	12	5	5	2	2	2
Pneumonia	11	4	4	2			
Influenza	2	3					
Asthma			1	1			
Bronchitis	1						
Whooping Cough	3	1					1
Tb. Contact		1					
T. & A.		1	1				
Unknown	4	2					

Upper respiratory disease is the etiological factor more often in the 2nd decade, but continues later in life than any other factor. Pneumonia is much more frequently the cause of bronchiectasis before the age of 10.

In considering the age at onset in relationship to the disease at onset, the cases which gave an upper respiratory etiology seemed to occur more frequently between the ages of 10 and 20. Also upper respiratory disease tends to continue as the causative factor later in life than any other disease. In contrast to this, pneumonia has its highest incidence in the first decade of life.

The next relationship, that of sex to etiology, shows another striking fact. The group of upper respiratory cases have a higher proportion of men than women, and conversely the cases of pneumonia alone, whooping cough, bronchitis, influenza, all seem to have a higher proportion of women. This fact is important only as it is another point of differentiation in etiology.

CHART V

Disease	Etiology and Sex	Male	Female
Upper respiratory infections	Head colds	0	0
	Sinusitis	0	0
	Head colds and pneumonia	20	12
Pneumonia		8	13
Influenza		1	4
Asthma		1	1
Bronchitis		0	1
Whooping Cough		1	4
T. & A.		0	2
Unknown		4	2

Upper respiratory infection is more frequently the etiology among males. Pneumonia, influenza and whooping cough are more frequently the etiology among females.

When we see that the upper respiratory group has age at onset and sex incidence characteristics that are different from those of the pulmonary group, it lends support to the belief that there is no one single cause of bronchiectasis. There seem to be several diseases that have etiological significance. As the cases with upper respiratory etiology tend to occur later in life than the pneumonia cases, I suggest that these form a group where repeated upper respiratory infections, either head colds or sinusitis, over a considerable period of time, have led to pulmonary infection. This would be a type of slow onset, where the mechanisms of inhalation or lymphatic drainage play their part.

The pneumonia group represents a type in which the bronchiectasis has occurred as the result of one acute infection. At the risk of repetition I would like to sum up the data so far presented. We have here a group of cases who have had bronchiectasis for a considerable period of time for the most part. They have acquired the pulmonary disease in the majority of cases before the age of 20. In fact, nearly a third of them developed the disease before the age of 10. They ascribe their bronchiectasis to several diseases. The greater number acquired it following an upper respiratory infection. However, 21 cases had pneumonia at the onset which was not preceded, so far as they know, by an upper respiratory infection. The upper respiratory group and the pneumonia group have special points of differentiation, namely, age at onset and sex which, to my mind, confirm the statements of the patients and indicate that bronchiectasis can occur without a preceding upper respiratory infection.

I will now take up the relationship of the etiology as given by the patients and the presence of sinusitis as shown by x-ray.

CHART VI.

RELATION OF DISEASE AT ONSET TO SINUSITIS (BY X-RAY)

Disease at Onset		Incidence of Sinusitis	
		Sinusitis	Negative
Upper respiratory infections			
Head colds	21 cases	11	10
Sinusitis	6 cases	6	0
Colds with pneumonia	5 cases	5	0
Total	32 cases	22	10
Pneumonia	21 cases	12	9
Influenza	5 cases	2	3
Asthma	2 cases	2	0
Bronchitis	1 case	0	1
Whooping Cough	5 cases	4	1
Tb. contact	1 case	0	1
T. & A.	2 cases	1	1
Unknown	6 cases	3	3
Total	75 cases	46	29

In the upper respiratory group the incidence of sinusitis is a little less than 69 per cent. As one would expect, it is greater than in the pneumonia group, but note that in this latter group 57 per cent have sinus infection.

It is surprising to note here the relatively high incidence of sinusitis as found in the group of 21 cases of pneumonia. Twelve have now a demonstrable sinus infection. Nine have a negative sinus x-ray. As one would expect, the group of "colds," "sinusitis," and "colds and pneumonia" have a much higher incidence of sinus infection. Twenty-two have sinusitis and 10 have no evidence of sinusitis. How can we explain the sinus infection in cases which do not give upper respiratory infection or sinusitis as an etiological factor?

It is striking to observe the high incidence of frequent upper respiratory infections, subsequent to the onset of bronchiectasis. Chart VII correlates the frequency of upper respiratory infection with x-ray evidence of sinusitis. The x-ray picture corresponds to the history of upper respiratory infections occurring subsequent to the onset of bronchiectasis rather than to the ascribed etiology.

Naturally it can be stated that there are grades of chronic sinusitis, and that there are cases with a negative sinus x-ray, who

have had repeated attacks of acute sinusitis with recovery. In other words, can we rely entirely on the negative x-ray evidence of sinusitis to rule out sinus infection etiologically? Or can bronchiectasis occur in a case with a negative sinus history and absolutely negative sinus examination?

CHART VII.

RELATIONSHIP OF SUBSEQUENT UPPER RESPIRATORY INFECTIONS AND SINUSITIS

A. Group as a Whole		
Upper Resp. Inf. Since Onset	Sinusitis	Negative
Slight	9 cases	17 cases
Frequent or Severe	37 cases	12 cases
	46 cases	29 cases
B. Comparison of Pneumonia Group and Upper Resp. Group		
1. Cases of Pneumonia		
Upper Resp. Inf. Since Onset	Sinusitis	Negative
Slight	2 cases	7 cases
Frequent or Severe	10 cases	2 cases
	12 cases	9 cases
2. Cases with Head Colds or Sinusitis at Onset		
Upper Resp. Inf. Since Onset	Sinusitis	Negative
Slight	2 cases	3 cases
Frequent or Severe	20 cases	7 cases
	22 cases	10 cases

The incidence of sinusitis closely parallels the frequency or severity of upper respiratory infections occurring since the onset of the bronchiectasis.

In this series of 75 cases, 46 now have chronic sinus infection and 29 have negative sinuses, according to the x-ray evidence. Of these 29 negative cases, 18 have a history or clinical evidence suggestive of sinusitis, either a recurrent acute sinusitis or a mild chronic sinusitis.

Eleven cases have absolutely no evidence of past or present sinusitis. In these negative cases the disease at onset is as follows:

Pneumonia, six cases.

Upper respiratory infection, one case. Here the infections were more pharyngeal than nasal.

Influenza, one case.

Whooping cough, one case.

Tb. contact, one case.

T. and A., one case.

In this small group, comprising 14½ per cent of this series, sinusitis seems to have played no part in the onset or subsequent course of their bronchiectasis.

In considering the group who give "head colds" as the etiological factor, it is evident that a negative sinus x-ray is not conclusive. In the pulmonary group we have another very important thing to consider, and that is the undeniable fact that these people had a severe pulmonary infection whether or not they had a nasal infection at the same time.

To sum up the facts so far presented, I will give very briefly the conclusions which I have reached at this point. There seems to be definitely several different diseases which can cause bronchiectasis. The largest group is that of upper respiratory infection. Pneumonia alone accounts for a considerable number, 21 cases in this series. This statement, however, does not give a clear picture of the situation when we see the patient in later years. As you have seen, these cases show a marked susceptibility to severe upper respiratory infection. As time goes on these repeated upper respiratory infections have produced the picture of sinus disease which is greater than the etiology would indicate. This point may seem quite obvious, but I have a feeling that when we see a case of bronchiectasis with sinus infection, there is a tendency to consider the sinusitis as the original cause of the bronchiectasis without considering the possibility that the sinusitis may have developed subsequently and independently of the pulmonary disease.

The next problem to be considered is the relationship of sinusitis to the pulmonary infection. Whether or not the sinus infection was an important factor at the onset of bronchiectasis, once the patient has acquired a sinus infection, does it predispose him to further pulmonary damage? Also, is there any correlation between the degree of sinusitis and the extent of bronchiectasis? The practical question arises, when considering the therapeutic management of one of these cases, whether we should consider a radical sinus operation. So far as the bronchiectatic cavity is concerned, we do not expect to eradicate it by sinus surgery. But the importance of removing the sinus infection is shown in the next chart, in which you may observe a high percentage of sinusitis in cases who have had repeated pulmonary infection.

In Chart VIII I have collected 16 cases who had a subsequent pulmonary infection. Two of these cases, as you see, have had three attacks of pneumonia and one has had two attacks of pneumonia occurring after the onset of bronchiectasis. Six of these cases had an onset attributed to head colds or sinusitis. In this group there is evidence of definite sinus infection. There are seven cases who attribute the onset to pneumonia. Three of these have no evidence of sinusitis. Four have a marked degree of sinus infection. The case of asthma and the two cases of whooping cough also have marked sinus infection. It seems that in these cases of recurrent pulmonary infection there is an extremely high incidence of severe sinusitis which constitutes a possible source of reinfection for the lungs.

CHART VIII.

RELATIONSHIP OF SINUSITIS TO REPEATED PULMONARY INFECTION

Case No.	Pulmonary Infection	Original Etiology	X-ray
1.	Empyema	Head Cold	Both ethmoids chronic c active infection, left frontal thickened
2.	Pneumonia	Sinusitis	Fluid both antra
3.	Pleurisy	Sinusitis	Chronic thickening both antra and ethmoids
4.	Pneumonia	Sinusitis	Both antra and ethmoids thickened
5.	Pneumonia	Head Colds	Neg. (clinically recur. ac. sinusitis
6.	Pneumonia 3 times	Head Colds	Left pansinusitis
7.	Pneumonia	Pneumonia	Negative
8.	Pleurisy	Pneumonia	Both ethmoids and antra pus
9.	Pneumonia 2 times	Pneumonia	Negative
10.	Pneumonia 3 times	Pneumonia	Negative
11.	Pneumonia	Pneumonia	Both ethmoids and antra secretion
12.	Pneumonia	Pneumonia	Both ethmoids and antra thickened
13.	Pneumonia	Pneumonia	Bilateral pansinusitis
14.	Pneumonia	Asthma	Both ethmoids and antra thickened; pus
15.	Pneumonia	Wh. Cough	Both ethmoids and antra thickened; pus
16.	Pneumonia and empyema	Wh. Cough	Both ethmoids and antra thickened; no pus

These 16 cases have had severe pulmonary infection subsequent to the onset of bronchiectasis. Four of these cases have negative sinus x-rays. The remaining 12 have a marked degree of sinusitis.

Chart IX gives the location of the sinus pathology. I cannot explain why no cases show infection of the right antrum or right ethmoid alone. In those cases where more than one sinus is involved, we find infection of the right antrum and ethmoid frequently. Does this bear any relationship to the fact that the left lower lobe of the lung is more often the site of bronchiectasis than any other one lobe? Does it suggest a homolateral factor?

In another chart, which I will show you presently, 20 cases of left lower lobe bronchiectasis are given, nine of which had x-ray evidence of sinusitis, and of these, two cases had right-sided sinusitis alone. Of six cases with right lower lobe bronchiectasis, two have sinusitis and both have more involvement of the left side than the right.

CHART IX

LOCATION OF SINUSITIS

Sinuses Involved	No. of Cases
Right antrum only	0
Left antrum only	4
Both antra	3
Left ethmoid only	3
Both ethmoids only	3
Right ethmoid only	0
Right frontal only	1
Left frontal only	0
Both ethmoids and antra	18
Both ethmoids and frontals	2
Right ethmoids and right frontal	1
Right pansinusitis and left antrum	1
Right pansinusitis and left antrum and ethmoid	1
Left pansinusitis	1
Left pansinusitis and right antrum and ethmoid	3
Both pansinusitis	5
	46

The ethmoids and antra are about equally subject to infection. In 18 cases there was bilateral infection of the ethmoids and antra.

But it is suggestive in examining the four cases of left antrum infection to find that they all have infection of the left lower lobe, two of them with a left lower lobe only. Three of these cases give a history of upper respiratory infection, and one of whooping cough at the onset of the bronchiectasis. One case was complicated by a tumor in the mediastinum and this case had an involvement of both

lower lobes and right middle lobe. The pathology in the sinuses of these four cases is very slight, as follows:

Case 1. Small amount of cystic membrane in the left antrum.

Case 2. Left antrum membrane very slightly thickened.

Case 3. Cyst in left antrum.

Case 4. Left antrum chronic. Small amount of fluid.

Case 4 is the patient with mediastinal tumor and extensive bronchiectasis.

Without giving each case in detail, we may summarize them as a whole in regard to the x-ray diagnosis. Seventeen cases showed pus or retained secretion in addition to changes in the mucous membrane in the sinuses affected. Three cases had cysts only. Six cases had very slight thickening of the lining membrane of the sinuses. Ten cases had moderate thickening, and the remaining nine cases had a marked thickening of the membrane. Generally speaking, those cases that had the greater number of sinuses involved, also had a more severe pathological change in the membrane.

I should like to call your attention to the high incidence of infection of both ethmoids and antra in this series. In fact, it is striking that the ethmoid was infected quite as frequently as the antrum. This is very important from the operative point of view. In those cases which have been operated on where the ethmoids and the antra were infected, the best results were obtained, as you would expect, where a thorough exenteration of the ethmoids was done, in addition to a radical antrum operation. In fact, no matter how bad the antrum seems to be, if the ethmoid is also infected, unsatisfactory results will be obtained if the ethmoid is only partly cleaned out.

In the next two charts I will show the relationship of the extent of bronchiectasis to the sinusitis. In the first chart I have gone into the question of the location of the sinusitis in cases of one lobe bronchiectasis. In the second chart is given the relationship of sinusitis to the location of bronchiectasis.

There is no evidence here that there is any connection between the side of the chest which is affected and the location of the sinusitis. The question of the incidence of sinusitis in cases involving one lobe is important to the thoracic surgeon as these are the cases which lend themselves more favorably to lobectomy. It is important to note that the incidence of sinusitis is much lower among these cases than it is where more than one lobe is infected.

CHART X.

SINUSITIS IN CASES OF UNILATERAL BRONCHIECTASIS

Lobe Involved	Incidence of Sinusitis		Sinuses Involved	
L. L. L. 20 cases	Sinuses neg.	11 cases	Left antrum	1 case
	Sinusitis	9 cases	Right frontal	1 case
			Both eth. and ant.	5 cases
			Both eth. and frontals	1 case
			Right frontal and right ethmoid	1 case
R. L. L. 6 cases	Sinuses neg.	4 cases	Left ethmoid	1 case
	Sinusitis	2 cases		
			Left frontal and ethmoid Both antra	1 case
R. L. L. and R. M. L. 5 cases	Sinuses neg.	2 cases	Both ethmoids	2 cases
	Sinusitis	3 cases	Both pansinuses	1 case
L. L. L. and L. U. L. 5 cases	Sinuses neg.	2 cases	Both eth. and ant.	2 cases
	Sinusitis	3 cases	Left eth.	1 case

The side on which the sinusitis occurs does not correspond to the side of the chest which is affected. Cases in which one lobe is infected have a lower incidence of sinusitis.

CHART XI.

RELATIONSHIP OF LOCATION OF BRONCHIECTASIS TO INCIDENCE OF SINUSITIS

	Sinusitis
1. L. L. L.	45%
2. L. L. L. and L. U. L.	60%
3. R. L. L.	33⅓%
4. R. L. L. and R. M. L.	60%
5. R. U. L., 1 case	Neg.
6. B. L. L.	67%
7. L. L. L. and R. M. L.	73%
8. R. L. L. and L. U. L., Situs Inversus, 1 Case	Sinusitis
9. B. L. L. and R. M. L.	67%
10. B. L. L. and L. U., 1 Case	Neg.
11. Pan Bronchiectasis	75%

Cases of bronchiectasis where one lobe only is affected have a lower incidence of sinusitis than those cases where more than one lobe is affected.

Chart XI gives the relationship of infection of the various lobes of the lung to the incidence of sinusitis. Cases in which one lobe only is involved have a lower percentage of chronic sinusitis than those in which two or more lobes are diseased. In other words, the prevalence

of sinus infection parallels the extent of the bronchiectasis. Reduced to percentages, the sinuses are infected in 40 7/10% of those cases in which one lobe only is involved, and in those with more than one lobe involved, in 73% of the cases.

CONCLUSIONS

1. I do not think that we can attribute all cases of bronchiectasis to a preceding upper respiratory infection although the greater number of cases certainly have had a high incidence of head colds at the onset.

2. Other diseases, pneumonia being the most important, can cause bronchiectasis.

3. When we see the patient in later years, we find a very high incidence of sinus infection. This is higher than can be accounted for by the history of the disease at onset. It bears a closer relationship to the history of subsequent upper respiratory infection.

4. The incidence of sinusitis corresponds to the extent of the bronchiectasis.

5. Once the patient has acquired a chronic sinusitis, the chance of further damage to the lungs is increased because these patients are more susceptible to repeated respiratory infections.

258 BEACON STREET.

XXX

II. CLINICAL EXPERIMENTS WITH REFERENCE TO THE INFLUENCE OF THE WATER METABOLISM ON THE EAR

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AND

Dida Dederding

Copenhagen

We have endeavored to demonstrate the influence of the water metabolism on the ear both by dehydration and by hyperhydration.

Already Politzer[1], by his well known pilocarpin treatment aimed at dehydration, pilocarpin giving rise to strong perspiration and salivation with an eventual subsequent improvement of audition. Hence P. Schubert[2], in 1892, drew attention to the fact that the improvement in hearing depended upon the patient's simultaneously being deprived of a sufficient quantity of liquid and becoming thirsty. He also described the peculiar initial contrary phase, in which the hearing temporarily decreases and subsequently increases, a phenomenon which we have likewise observed after administration of pilocarpin (Table I) as well as after application of a series of other dehydrating processes. (3.4.) This holds good, for instance, for such diuretics as agurin, diuretin and euphyllin, the latter of which is particularly efficient in producing a very considerable improvement in hearing. (Table II.)

However, the most distinct effect was produced by the very efficient mercury preparation *salyrgan* (mersalyl), which rapidly produces profuse diuresis. We have previously published 77 experiments carried out on 46 patients. In 33 experiments an indisputable improvement in hearing was obtained. Among these we have selected the following instances. (Tables III, IV, V.)

It is evident that the effect always depends upon the patient's obtaining loss of weight and thirst. Salyrgan also produces an initial contrary phase, which is conjoined with salivation, whereas the subsequent improvement in hearing frequently does not occur before the increase in diuresis is about to cease. In some cases the improvement in hearing is accompanied by a spontaneous nystagmus. The partial

relief from an existing headache has also been witnessed. The improvement in hearing just as with spontaneous improvement, has the character of improved sound conduction and not of improved sound perception. As to bass deafness we have witnessed maximal rises from 150 d. v. to 55 d. v. as to whispering voice from $\frac{\text{ad aurem}}{0,50\text{ m.}}$ to $\frac{14,00\text{-}20,00\text{ m.}}{20,00\text{ m.}}$, and as to conversation voice from $\frac{0,10\text{ m.}}{0,25\text{ m.}}$ to $\frac{20,00\text{ m.}}{20,00\text{ m.}}$

Whereas the increase of diuresis after salyrgan in normal individuals is followed by a compensatory decrease, this is not the case in Ménière patients. This is suggestive of their having abnormal water depots.

As a contrast with these results we have carried out a series of hyperhydration experiments. We made use of a water test, in which the fasting patients were requested to drink 1000 cc. of water early in the morning. Beforehand the weight was determined and the bladder was emptied. After the lapse of four hours the weight was once more determined and the total diuresis was measured. A careful function test was performed prior to, as well as several times during, the experiment. In a great number of cases of Ménière's disease the patients were found to incur more or less pronounced reduction in hearing during such a water test. This acoustic reaction, just as that in the cases of spontaneous deafness, has the character of a sound conduction affection with distinct bass deafness. Moreover, there is a shortening of air and bone conduction for a_1, and the hearing for whispering is reduced, eventually for conversational voice also. In a great number of cases, concurrently with examination of hearing we determined the fluctuations in the concentration of the blood, with the help of a hematocrite, for during the water test a dilution of blood occurs which, in most cases, runs a somewhat irregular course as regards time and intensity.

The most interesting feature is, however, that this blood concentration curve (dotted line) and the acoustic reaction curve (plain for a_1 air conduction) in by far the majority of cases (77 out of 97) run fairly parallel, as is seen in Charts I, II, III and IV.* In several cases the maximal hydremia is not only accompanied by the maximal acoustic reaction but also by a distinct vestibular reaction in the form of a spontaneous nystagmus (Charts III and IV), and in some cases, besides by buzzing in the ears, and by the peculiar sensation of pressure in the ear (Chart VI), which is characteristic of spontaneous

*Time of testing indicated horizontally, divisions of hematocrite and seconds of vibration period of tuning fork heard indicated vertically. One liter of water given immediately after first test.

exacerbation. It seems as though the progress of the blood concentration curve of Ménière patients were more irregular, with greater fluctuations, than that of the normal.

Control experiments performed on otosclerosis patients yielded no acoustic reaction, which indicates that the reaction depends upon a sufficiently mobile stapes. In this respect some control experiments performed on two colleagues are very illustrative. One of them had previously had a transitory right-sided middle ear catarrh and, as a child, a suppuration of the left ear, which had resulted in permanent partial deafness. This evidently is due to a stapes ankylosis, for during the water test (Chart V) the reduced hearing on the left side persisted unchanged, whereas a distinct reaction corresponding to the hematocrite curve was recorded for the also reduced hearing on the other ear.

The other apparently normal person experienced not only a marked acoustic reaction on both ears corresponding to the deepest fall of the blood curve (Chart VI), but also an increasing sensation of pressure in the ear. On questioning him we learned that he had previously experienced giddiness after tobacco smoking and that his grandmother had suffered from typical attacks of Ménière's disease.

Also the experiments in hyperhydration give rise to abnormal general reaction. Several patients during the water test complain of headache and nausea, and about half of all patients undergo an increase in weight amounting to 250 gm. or more in the course of the four hours, whereas a loss or an increase of weight of not more than 150 gm. is observed in the control experiments on normal individuals. Several times we have observed a water retention of 600-800 g. Some few patients are not at all able to resorb the large quantity of water and immediately react by vomiting and profuse diarrhea.

The administration of water as a clinical test moreover has the drawback that it depends upon the subject's preceding diet. A diet poor in liquid or rich in salt may give rise to a retention which is not pathological. Therefore it is important to ascertain that the patients have not brought themselves into such a spurious situation. Thus we always examine whether the patient prior to the experiment has felt thirsty and whether his tongue is dry or moist. In case of retention the experiment is repeated, the test subject this time previously receiving a liberal supply of liquid, eventually being placed in a recumbent posture, which normally causes a much greater discharge and, consequently, a loss of weight. We have observed that some patients during good periods have a normal discharge, and during bad periods retention.

However, we have recently given the patients 800 gm. of water only, administered in four portions of 200 gm. each at interims of one-half hour, as indicated by Labbé and Violle.[5] This is not so unpleasant to the patient, but the acoustic and vestibular reactions do not become so marked either.

It should be noted that acoustic and vestibular reactions may very well be observed without simultaneous universal retention of water. As will be seen, the result of these experiments correspond to those obtained by Fürstenberg, Lashmet and Lathrop.[6] By hyperdosation of water Fürstenberg et al. in a Ménière patient were able to provoke giddiness and nystagmus with simultaneous increase of weight. However, as they obtained the same result by administration of sodium chloride, which moreover was accompanied by a loss of weight, they concluded that the retention of sodium and not the retention of water was the primary factor. These investigations were carried out with admirable care and accuracy. However, if Fürstenberg and his collaborators come to another conclusion than we, this is due to their not having been aware of the fact that giddiness and nystagmus do not only occur in case of exacerbation of the condition but also in case of improvement. The chief thing is to know whether the hearing increases or decreases. Therefore it is the auditory condition which shall show whether a treatment is adequate or not. Nor are Fürstenberg's results of his treatment quite convincing as long as we know nothing about its influence on audition. It is true that, a priori, it is extremely probable that his diet poor in salt is beneficial in case of Ménière's disease because it is dehydrating, as is also the diet of Földes[7], with the fairly restricted intake of fluid.

About 10 years ago we also carried out a series of experiments of hyperdosation with sodium chloride, which certainly are far from being so accurate as Fürstenberg's experiments, but we have at the same time determined the hearing. Unfortunately our records of those experiments have been lost, and therefore we have not mentioned them before. We are, however, able to assert that, in isolated cases, a temporary improvement of hearing ensued when the dosage of sodium chloride gradually increased and the quantity of water administered was so small that the patients became thirsty. For as long as a surplus of salt is given, water is drawn from the tissues into the blood-stream, the sodium chloride simultaneously going the opposite way, as seen by the simultaneous dilution of the blood determined by hemoglobin test. Finally, however, a balance between tissue and blood-stream is reached, and one cannot get any further without evacuating water and salt with the help of an efficient diu-

retic. In isolated cases we have done this and witnessed a violent effect on diuresis and hearing, but it is a treatment exceedingly unpleasant for the patient.

Accordingly we must conclude that the essential factor in Ménière's disease is a propensity to water retention, partly universal, partly local, above all in th

In a following communication* we shall endeavor to supplement these clinical observations by the results derived from a series of experiments.

TABLE I

	Upper limit	Lower limit	a1 Bone (norm. 15″)	Whisper	Spont. Nyst.
Before inject.	16000 d. v.	40 d. v.	0″	0,40 m.	
	16000 d. v.	30 d. v.	0″	10,00 m.	
½ hr. after inject. of Sol. chl. pilocarp. 2% 0.6 ccm.	15000 d. v.	90 d. v.	0″	0,50-0,80 m.	Rot. to L. Hor. to L.
	14000 d. v.	40 d. v.	0″	1,00-2,00 m.	
2 h. after inject.	16500 d. v.	55 d. v.	0″	0,50-1,00 m.	
	15000 d. v.	26 d. v.	0″	3,00 m.	
5 h. after inject	17000 d. v.	32 d. v.	2″	1,20 m.	
	15000 d. v.	26 d. v.	0″	hardly 20,00 m.	

TABLE II

	Upper limit	Lower limit	a1 Bone (norm. 15″)	a1 Air (norm. 60″)	Whisper
Before	14000 d. v.	24 d. v.	9″	22″	10,00 m.
	14,000 d. v.	24 d. v.	4″	38″	10,00 m.
3 h. after Euphyllin ctgr. 30	15000 d. v.	90 d. v.	0″	5″	0,0 m.
	14000 d. v.	>150 d. v.	0″	5″	0,0 m.
24 h. after					10,00 m.
					10,00 m.

*To appear in an early issue.

TABLE III

7/6	Upper limit	Lower limit	at Bone (norm. 15″)	at Air (norm. 40″)	Whisper	Weight
Before inject.	14000 d. v.	36 d. v.	5″	22″	3,00 m.	69,7
	8000 d. v.	150 d. v.	2″	12″	0 m.	
24 h. after inject. of Salyrgan 10% 1 ccm.	13500 d. v.	24 d. v.	6″	21″	7,00 m.	
	8500 d. v.	65 d. v.	5″	18″	ad aurum	68,7
31 h. after inject.	14000 d. v.	26 d. v.	7″	21″	7,00 m.	
	8000 d. v.	70 d. v.	6″	15″	0,05 m.	
48 h. after inject.	16000 d. v.	28 d. v.	6″	17″	12,00 m.	68,7
	8500 d. v.	70 d. v.	6″	14″	0,05 m.	

	6/6	7/6	8/6
Fluid intake during 24 hours	600	600	600
Diuresis	650	1250	550
Specif. weight	1024	1013	1024

TABLE IV

28/8	Upper limit	Lower limit	at Bone (norm. 15″)	at Air (norm. 60″)	Whisper
Before inject.	14000 d. v.	80 d. v.	0″	5″	aa-0,25 m.
	14000 d. v.	38 d. v.	0″	20″	0,5-4,00 m.
8 h. after inject. of Salyrgan 10% 1 ccm.	14000 d. v.	38 d. v.	0″	12″	0,20-12,00 m.
	14000 d. v.	30 d. v.	10″	33″	20,00 m.
11 h. after inject.	14000 d. v.	24 d. v.	8″	15″	14,00-20,00 m.
	14000 d. v.	24 d. v.	10″	33″	20,00 m.
22 h. after inject.	14000 d. v.	40 d. v.	0″	12″	14,00-20,00 m.
	14000 d. v.	36 d. v.	0″	30″	10,00-20,00 m.

	27/8	28/8	29/8
Fluid intake during 24 hours	800	600	600
Diuresis	600	1560	New Salyrgan injection
Specif. weight	1030	1013	

TABLE V

7/7	Upper limit	Lower limit	a1 Bone (norm. 15″)	a1 Air (norm. 40″)	Whisper	Weight
Before inject.	11000 d. v. / 12000 d. v.	22 d. v. / 24 d. v.	9″ / 7″	13″ / 11″	3,00 m. / 1,5-2,00 m.	65,7
6 h. after inject. 1 ccm. 10% Sol. Salyrgan	11000 d. v. / 13000 d. v.	22 d. v. / 18 d. v.	11″ / 9″	36″ / 17″	2,0-5,0 m. / 2,00 m.	65,2
46 h. after inject.	9000 d. v. / 12000 d. v.	19 d. v. / 16 d. v.	11″ / 11″	37″ / 27″	15,00 m. / 15,00 m.	64,0

	6/7	7/7	8/7
Fluid intake during 24 hours	1000	500	500
Diuresis		1300	375
Specif. weight		1014	1027

CHART I

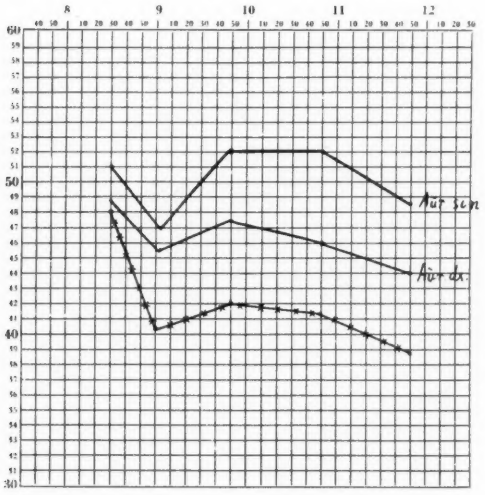


CHART II

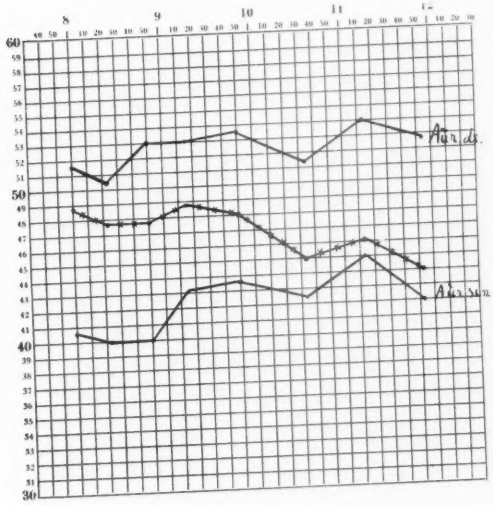


CHART III

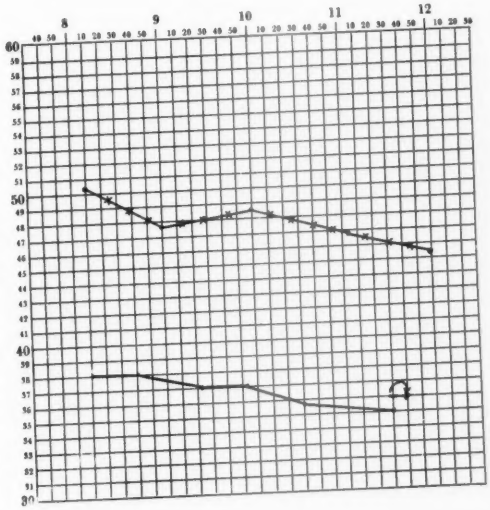


CHART IV

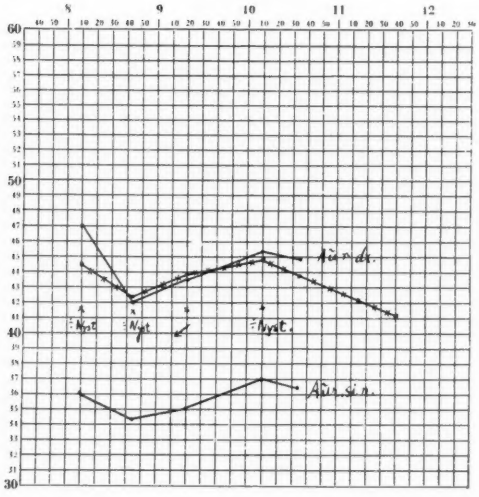


CHART V

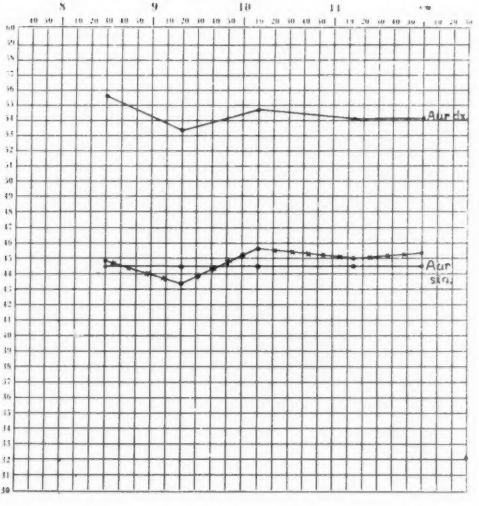
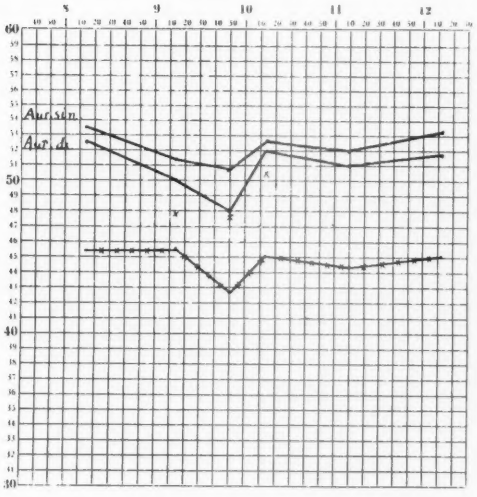


CHART VI



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XXXI

PRIMARY BILATERAL SEROUS ORBITAL CELLULITIS*

ABRAHAM FINE, M.D.

BROOKLYN

It is generally conceded that diseases of the orbit fall within the domain of the nose and throat specialist. In the past, suppurative lesions of the orbit were treated by the ophthalmologist, who felt that he had done all that was necessary when he made an incision into the orbit for the purpose of drainage. But we know today that such therapy is inadequate. If the sinuses are the etiologic agent, as they so frequently are, drainage of the sinuses by the rhinologist is required, in addition to treatment of the orbital complication. The field of otolaryngology is constantly expanding, and the alert specialist is often compelled to explore the neighboring specialties.

The subject I am presenting deals with serous orbital cellulitis and, in particular, with a rare form to which I have applied the name "primary bilateral serous orbital cellulitis." The condition has never before been described in the nose and throat literature. References to it appear in the journals of ophthalmology under the heading of "serous tenonitis." This latter description, in my opinion, is based on a misconception of the anatomy and pathology of the disease, which, according to ophthalmologists, is confined to Tenon's capsule. It is my purpose to show that this disease involves all the structures of the orbit. It is a true serous orbital cellulitis, and not merely a tenonitis.

I wish to emphasize that this disease has a practical significance for the otolaryngologist. Imagine being called to treat a patient complaining of excruciating pain over the frontal and maxillary sinuses, with an apparent orbital complication, and being compelled, as a last resort, to operate on what eventually turns out to be normal sinuses. Such was the unfortunate lot of the writer. The disease, in addition, has a protean character, which lends itself to confusion with a variety of conditions, which it closely simulates.

*Presented before the Nose and Throat Section of The Kings County Medical Society, February, 1937.

From the Nose and Throat Service of the Coney Island Hospital, Dr. T. B. Wood, Director.

The following case report presents the characteristic features of this unusual clinical entity.

REPORT OF A CASE

CASE 1.—A young negress, age 23, was admitted to the nose and throat service of Dr. Thomas B. Wood at the Coney Island Hospital on September 4th, 1936. She complained of severe pain in and above the right eye, of five days' duration. There was no antecedent illness, upper respiratory infection, or history of trauma. She never before had any similar attacks. Except for gonorrhea contracted six years ago, she had always enjoyed good health. An examination made by the writer on the day of admission showed the following salient points: "Patient in extreme pain; slight edema of the right upper lid, bulbar conjunctiva diffusely congested, but no localized swelling present; pupils react to light; ocular motion free in all directions except on looking up; marked tenderness over the right frontal sinus and slight tenderness over the maxillary and lacrimal bones. The left eye is normal. The mucosa of the nose appears normal, and there is no discharge; slight congestion of the fauces; no infection of the face." The physical examination by the house officer revealed no abnormal findings outside of the local condition. My provisional diagnosis was "acute frontal and ethmoid sinusitis, with beginning orbital involvement."

On the following day, the local condition was definitely worse. There was marked edema of the lids, moderate chemosis and extreme restriction of motion in all directions. Tenderness was marked over the right frontal and maxillary sinuses. There was a small tender right pre-auricular gland. The temperature was normal (and, in fact, remained so throughout the course, except for a rise to 101 degrees on two occasions, following a milk injection of 10 cc.). The W. B. C. count was 9,100; polys. 76 per cent, lymphos. 24 per cent; H. B. 77 per cent and R. B. C. 3,800,000. The x-ray report at this time stated that with the exception of slight infiltration of the right ethmoid sinus, the sinuses were essentially negative. On the same day, faced by an orbital condition growing progressively worse, and harrassed by an absence of local pathology to explain it, except for a questionable sinus involvement, I decided to do an intranasal exploratory operation on the right sinuses. Puncture and irrigation of the right maxillary sinus revealed no pus. The ethmoidal cells were opened, cell by cell, and appeared normal. The sphenoid ostium was enlarged and no pus was found. I encountered difficulty in probing the frontal sinus, but in view of the negative findings on the other sinuses, made no further attempts.

There was a definite improvement following the operation, but a relapse to the original condition set in soon. This alternate improvement and retrogression which occurred frequently in the course is characteristic of the disease. On September 11th (one week after admission), the left eye became involved. It went through the same evolution as the right eye. At the height of the condition, the patient presented an unhappy picture. She was in severe pain. Both eyes were closed, and a watery discharge trickled over the lids. On raising the lids, the bulbar conjunctiva was seen markedly edematous, particularly in the lower fornix, which protruded between the lids. Exophthalmos was moderate. The eyeballs were very tender to the touch, and practically motionless. The media were clear and the fundus, except for a slight distension of the veins, was normal. Vision was 20/30 in both eyes. At this stage, the picture resembled an aseptic cavernous

sinus thrombosis, except that the signs of venous stasis in the lids, conjunctiva and retina were absent. Consultations with the members of the medical and neurological departments revealed no general disturbances. The dental consultant found no evidence of focal infection. The Wassermann test was negative and remained uninfluenced by provocative salvarsan. Blood culture was negative; urine negative; complement fixation test was positive for gonorrhea.

To throw some light on the precise nature of the inflammatory condition, I inserted a two-inch needle of large gauge through the skin of the right lower lid, under aseptic conditions, and passed it along the floor toward the apex of the orbit. I succeeded in withdrawing from behind the eyeball a few mm. of clear, straw-colored fluid, which was cultured. The report was "albumin negative, globulin 2+, specific gravity 1.015; smear, no organisms seen; culture, staphylococcus albus (probable contamination)." Several days later I repeated the procedure on the left orbit. Failing to aspirate free fluid, I injected about 2 cc. of sterile saline into the orbital contents and recovered it in my syringe. A broth culture was made. The report on this was "smear—no organisms seen; culture—no growth."

On the 23rd of September the picture presented some of the aspects of an intracranial lesion. The patient had projectile vomiting on two occasions. The head pains were so severe that she sat holding her head and was unable to sleep. The pain could only partially be relieved by morphine. These symptoms occurred while the local condition was definitely improving. A lumbar tap taken previously showed the fluid to be clear, pressure 10 mm. mercury, glucose and chloride determinations within normal limits; cell count, many R. B. C.; culture, no growth. A second tap taken on the 24th showed no evidence of an increased intracranial pressure. The fluid remained clear and the other findings were normal.

Impairment of vision in the right eye soon became an outstanding complaint. It was now 20/200 (20/30 previously). I could find no explanation for it in examination of the media and retina. Using a 2 mm. size colored test object, I examined each eye for central scotoma. There was no difficulty in recognizing colors with the left eye, but in the right eye there was a definite relative central scotoma, which indicated a retrobulbar neuritis. The peripheral fields of vision, tested by the confrontation method, were negative.

Six weeks after admission, the patient was discharged. The vision of the right eye had returned to normal. The edema was gone and ocular movements were normal. The patient was comfortable, and remained so for about a month, while under observation.

ETIOLOGY

In a condition such as this, involving both eyes, and not attended by fever or leucocytosis, the answer to the cause is to be found in a study of the individual's constitution. In the literature, rheumatism, gout, influenza and refrigeration have been incriminated. Syphilis plays an unimportant part. No organisms have ever been isolated in the tissues or in the serous exudate. In the particular patient under study, no local or constitutional pathology was demonstrable. The pain and tenderness over the frontal and maxillary sinuses is due

to referred pain from involvement of the fifth nerve. The sinuses could definitely be ruled out. The nasal examination, exploratory operation on the sinuses, the culture taken from the maxillary sinus by injection of normal saline and the recovery of the same in the syringe, were all negative. Lipiodol studies of the right antrum showed no thickening of the mucosa. The slight infiltration of the right ethmoid reported in an otherwise negative x-ray, was probably the result of a previous upper respiratory infection. The possibility of an allergic basis was suggested by Dr. Wood. A search of the literature revealed that no investigation had ever been made along this line. Accordingly, Dr. Sherman of the allergy department was requested to make a study. His report was that the eye findings in this case were very unusual and atypical, as far as pollen sensitivity was concerned. They were also unusual for contact dermatitis or conjunctivitis, as both ordinarily clear up in two or three weeks, after removal from the environment-containing excitant. All pollens, inhalants and food reactions were entirely negative. There was nothing in the personal or family history to indicate allergy. The blood differential study never showed more than 1 per cent eosinophilia. The writer removed a small piece of tissue from the chemotic conjunctiva of the lower cul de sac of the right eye for examination. The microscopic report stated "specimen shows an occasionally polymorpholeucocyte, lymphocytes and several plasma cells. In addition, there are several large cells which appear to have a vacuolated cytoplasm. Diagnosis—orbital tissue with low-grade inflammatory reaction." Apparently all the laboratory and clinical studies did not substantiate an hypothesis of allergy.

DIFFERENTIAL DIAGNOSIS

The following conditions will be briefly considered:

1. Septic and aseptic cavernous sinus thrombosis. In the septic type with high temperature, leucocytosis and pyemic manifestations present, there is usually no diagnostic problem, but greater difficulty arises in the aseptic type. The latter is a definite clinical entity. Dr. George C. Kreutz[1] recently described a case. The three characteristic findings in this condition are: (1) Marked venous stasis in the lids, conjunctiva and retinal vessels, associated with exophthalmos; (2) paralysis of the ocular muscles; (3) absence of temperature and of leucocytosis. In the case which I am reporting, there was no evidence of venous stasis and no true muscular paralysis.

2. Metastatic orbital cellulitis. This is usually unilateral, and commonly associated with pyemia and a positive blood culture.

3. Pulsating exophthalmos. In this condition the patient complains of head noises, and a bruit can be distinctly heard over the eye. A pulsation is transmitted to the finger, on making pressure over the eyeball, and marked evidence of venous stasis is present.

4. Panophthalmitis is almost always unilateral and associated with severe loss of vision. The media of the eye show destructive changes.

5. Orbital cellulitis is usually unilateral, the result frequently of a local pathological condition such as sinusitis, erysipelas, facial or dental infection, and is attended by leucocytosis and moderate to high temperature.

6. Primary serous tenonitis. I shall endeavor to show that cases similar to the one I have described, and captioned "primary serous tenonitis" in the ophthalmic literature, are, in reality, serous lesions of the orbital structures. My evidence will be threefold:

A. *Anatomical:* The orbit contains, in addition to the globe, which occupies the anterior part of the orbit, fat, muscles, vessels, nerves, connective tissue and lymph spaces. Tenon's fascia is a fibrous structure which envelopes the posterior two-thirds of the globe and is continuous with the sheaths of the muscles which pierce it. It also blends with the dural covering of the optic nerve and is continuous with the so-called periosteum of the orbit. For a long time Tenon's capsule was thought to be composed of a lymph sac with an endothelial lining, in which movement of the eyeball occurs as a ball in a socket joint. Inflammation, hence, would take the form of a teno-synovitis. Whitnall's[2] recent and thorough text on the anatomy of the human orbit gives us a fresh conception of this capsule and compels us to view lesions of it in a new light:

> "The capsule of Tenon is a thin envelope of connective and elastic tissue completely surrounding the eyeball from the circumference of the cornea in front to the entrance of the optic nerve behind; it is so closely applied to the globe, that in sections of the orbital contents it is not readily identified, but by pulling the structures apart posteriorly, a space between it and the globe, the episcleral space, can be formed. Careful scrutiny will show, however, that the interval it really occupied by a felting of extremely fine connective tissue comparable to the arachnoidea of the brain, though finer and denser. There is no sign of an endothelial lining, and the second or visceral layer of the capsule, as sometimes described, is merely that part of the adventitia which remains adherent to the sclera after separation of the capsule. The eyeball can move within the capsule, the adventitia being lax enough to allow of slight excursion, but in wider movements both globe and capsule move together as a whole upon the bed of orbital fat which is loosely connected to the capsule behind."

B. *Pathological:* Information concerning the pathology of this condition is very meager. Almost all of the cases fortunately go on to recovery and opportunity is not afforded to study the orbital structures. However, in a case of serous tenonitis reported by Benedict,[3] there was a complicating ulcer of the cornea, which required enucleation. The microscopic study was as follows: "The bit of Tenon's capsule excised was greatly thickened and the connective tissue fibers were very dense and compact; a piece of an extra-ocular muscle was included in the section; the fiber bundles were widely separated as though by edema, and marked degenerative changes were evidenced by great irregularity in straining and loss of striation." Unfortunately, retrobulbar tissue other than muscle was not included in the study. To throw light on what happens to the orbital tissues in cases closely resembling the one described, I searched the literature and found an article by Martin Cohen,[1] in which he describes several cases of nonsuppurative inflammation of the orbit, bilateral and afebrile, existing for several months and associated with pronounced exophthalmos. The clinical picture he presents, though not identical with that exhibited by my patient, bears such a close resemblance, that the difference appears to be more one of degree than one of kind. In performing a Kronlein operation, which was required to relieve the retrobulbar pressure, he found in the orbital cavity, indurated tissue (muscle, fat and connective tissue), the microscopic analysis of which demonstrated an actual diffuse infiltration by round cells. He says, "This inflammatory form of exophthalmos is the result of an inflammatory reaction in the retrobulbar tissue."

C. *Clinical*: It is pertinent to ask what happens when fluid accumulates in Tenon's fascia. Does it cause exophthalmos, or edema of the bulbar conjunctiva and lids, or both? The experiments of Charpy[5] (1911), throw light on this point. He studied the relations between the eyelids and the orbit by the injections of colored gelatine between the various muscle and fascial planes in the orbit. His results may be summarized as follows:

(a) Injections within the capsule of Tenon can distend and are confined to the whole episcleral space, from the corneal margin to the optic nerve. They produce anteriorly a regularly disposed tumefaction around and limited by the cornea, and lying beneath the bulbar conjunctiva and the anterior part of the capsule.

(b) Injections into the central part of the orbital fat form a retro-ocular mass, which is confined within the muscle cone in front; hence, moderate invasions of fluid into the central region neither in-

vade the conjunctiva nor reach the lids, but produce an exophthalmos and immobility of the globe.

(c) Injections into the peripheral part of the orbital fat, outside the cone of muscles, are chiefly confined to the lower part of the orbit, and lie between the orbital fat and the periosteum lining the orbital walls. They can reach both the conjunctiva at the fornices, and the eyelids behind the septum orbitale, by passing through any of the adipose orifices left between the attachment of the fascial sheaths of the muscles to the orbital walls, and this is the ordinary path along which fluids pass from the depths of the orbit to the eyelids.

Pathologic observations support the results of these experimental injections, and emphysemata, abscesses and hemorrhages have been shown to follow the paths outlined above.

With these facts in mind, demonstrating that fluid deposits in Tenon's fascia produce only chemosis and not exophthalmos, and that proptosis and immobility of the globe can be caused only by retrobulbar accumulations, let us critically examine the case reports in the ophthalmic literature described as "serous tenonitis." Von Gronholm and Hanna Golitzki-Olin[6] report an exophthalmos of 21 mm. in one eye and 20 mm. in the other eye. R. J. Sisson[7] describes in his patient a proptosis of 23 mm. in the right eye and 28 mm. in the left. These readings were recorded accurately with Hertel's exophthalmometer. E. DeLord[8] says, "The pronounced exophthalmos, the severe pain, the complete fixity of the eyeballs, the total ptosis and the chemosis, seemed to indicate a phlegmon of the orbit." In the mind of the last mentioned observer, there appeared to be a growing doubt as to the generally accepted nature of the pathology in his case of "serous tenonitis." Yet he did not forcefully carry his observations to their natural conclusion.

PATHOGENESIS

My conception of the development of the lesion, based on a careful study of the progress of the picture as it unfolded itself in the case described, as well as upon a close scrutiny of reported cases, indicates that the onset occurs in the muscles, primarily. This was indicated in my patient by the definite limitation of motion on looking up, long before any chemosis or exophthalmos had appeared. The next stage, which follows quite rapidly, is the extension of the inflammation from the muscles, by direct continuity along the fascial sheaths, to involve Tenon's fascia. This clinically manifests itself by

the appearance of chemosis and lid edema. The final stage is the involvement of the cellulo-fatty tissue of the retrobulbar space, with the resultant occurrence of exophthalmos, the degree depending on the extent of this involvement. These stages blend gradually, but can be clearly distinguished.

COURSE AND PROGNOSIS

The duration of the disease varies from several weeks to several months. The outlook is favorable and, as a rule, it tends to a spontaneous resolution. In the majority of cases no serious complications occur. However, in one reported case (Benedict), a perforating ulcer of the cornea required enucleation; in another, reported by Pincus,[11] there was a simultaneous involvement of the lacrimal and salivary glands. Von Gronholm described a residual ocular paralysis with diplopia, but his case occurred in a physician who had tabes, and it was quite likely that this condition played an important part in the muscle paralysis. Perhaps the commonest complication noted is an impairment of vision which ultimately clears up. In these cases the media and fundus are described in the literature as being normal, but, unfortunately, no further investigations were carried out to determine the cause of this acquired amblyopia. My studies revealed that the lesion was due to a retrobulbar neuritis.

TREATMENT

Based on the theory that most cases are the result of rheumatism, salicylates in large doses have found common vogue. Some writers have advised the use of forced fluids, but the dehydration treatment, I believe, is preferable. The latter was advised by Dr. Wood, and consists of 50 cc. of intravenous 50 per cent glucose, in addition to magnesium sulphate given by mouth and intravenously, and limitation of fluid intake to about 800 cc. daily. Hot compresses help to relieve the ocular inflammation, and plenty of ointment should be applied to the exposed conjunctiva. Puncture of the chemotic conjunctiva, which is so often done, is not advisable. It increases the inflammation and the risk of secondary infection.

SUMMARY

1. The characteristic findings of this unusual disease are the sudden onset with severe ocular pain, tenderness over the frontal and maxillary sinuses, edema of the lids and conjunctiva, with moderate to extreme exophthalmos and immobility of the ocular muscles. Fever and constitutional disturbances are absent.

2. This condition has never before received consideration from the otolaryngologist. It is a form of orbital pathology which unmistakably lies in his province.

3. An attempt was made in this study to throw light upon an obscure etiology. Two conditions received especial consideration; one, a possible sinus involvement, and the other, an allergic basis. Both, however, proved to be absent.

4. The writer wishes to suggest a re-classification of terms: "Serous tenonitis" to be reserved for those cases where only a localized swelling is present over an ocular muscle, and "primary serous orbital cellulitis" for cases similar to the one reported, where all the orbital structures are involved. The latter description would be more in harmony with anatomic, pathologic and clinical findings.

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XXXII

MASTOIDITIS CAUSED BY FRIEDLANDER'S BACILLUS*

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Infection with the *bacillus mucosus capsulatus* (Friedländer) may present a clinical picture exactly like that resulting from the well known streptococcus mucosus capsulatus: an insidious onset with merely fullness in the ear, mild tinnitus, and slight decrease in hearing; without pain or fever. The symptoms and findings at the onset suggest a simple secretory catarrh of the middle ear or a tubo-tympanic involvment. The mastoid process is invaded and early destruction of the deep bone structures occurs without the usual clinical findings of a coalescent mastoiditis. Hence what may be considered as a mild otitis media and mastoiditis can suddenly develop serious complications. It should be remembered, that although the majority of otologists, including myself, speak of the *streptococcus mucosus capsulatus*, bacteriologists state that it is actually a *pneumococcus*. However, the term "streptococcus mucosus capsulatus" relating to suppurative otitis media and mastoiditis has been established long ago by Neumann[1] and Ruttin[2] and continues to appear in the literature, especially the German, and in text books.[3]

The terms *streptococcus mucosus capsulatus*, *pneumococcus mucosus capsulatus* and *pneumococcus* type III, refer to the same organism. Although the coccus is gram positive and the Friedländer bacillus gram negative, both of these encapsulated organisms present similar cultural characteristics which may account for a similar clinical picture. A significant feature of both organisms is the production of a sticky mucoid colony on culture media, and a sticky mucoid reaction in the involved area of the patient, notably the bronchial tubes and alveoli of the lungs and the middle ear and mastoid cells. A pneumonia due to either of these organisms has a similar virulent course and high mortality caused not merely by the toxicity of the organism but also by the mechanical suffocation resulting from this

*Presented before the Western Section of the American Laryngological, Rhinological and Otological Society, Santa Barbara, January 29, 1938.

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sticky mucus. Bullowa[4] and his colleagues report the following: "In the 10 patients who came to necropsy the predominant lesion was a massive lobar consolidation, the gray-yellow granular lung containing a viscid gelatinous exudate. . . . Similar aputrid necroses were noted by us as well as by others in pneumococcic lobar pneumonia, particularly in pneumonia caused by pneumococcus type III." One naturally wonders why a pneumonia due to a pneumococcus type III or a Friedländer's bacillus should follow such a fulminating course, whereas when the middle ear and mastoid are invaded by either one of these organisms there is usually an insidious onset. The answer must be found in the highly potent local tissue reaction, which at once is devastating to lung tissue, whereas in the mastoid process this local tissue reaction causes a destruction of bone, the serious phase appearing after this has occurred. Solomon in an article in the Journal of the American Medical Association of March 20, 1937, entitled "Primary Friedländer Pneumonia," states: "It seems logical to assume from the foregoing that, in pneumonia due to Friendländer's bacillus, bacteremia apparently does not play the major rôle in the cause of death. This is in contrast to pneumococcus pneumonia in which the mortality rate varies directly as the incidence and severity of bacteremia. The cause of death in Friedländer's pneumonia is obscure in the absence of complications. . . . It is a fair assumption that death is due in large part to a severe general toxemia combined with intense pulmonary reaction to the organism."

B. MUCOSUS CAPSULATUS

The group characteristics of B. Friedländeri are as follows: short, non-sporing, capsulated, gram negative rods, giving a profuse mucoid growth on solid media, and usually fermenting carbohydrates with the production of acid and gas. Frisch in 1882 isolated a capsulated bacillus from patients with rhinoscleroma. In 1883 Friedländer cultivated a similar organism from the lungs of patients who died of pneumonia. Loewenberg in 1894 and Abel in 1896 cultivated similar organisms from the nasal secretions of patients with ozena. Besides these several other encapsulated bacilli have been described. Recent work, especially in America, by Avery,[5] Heidelberger,[6] and Julianelle,[7] had added a great deal to the differentiation of the types of B. mucosus capsulatus. They find that the immunological reactions of the Friedländer group are similar to those of the pneumococci, depending on the presence in the cell of two entirely different factors—a polysaccharide in the capsule, responsible for the type specificity, and a nucleo-protein in the organism responsible for the species-specificity. Julianelle recognized A, B, and C

types and an X-group of unclassified strains. This nomenclature was used by Bullowa in the identification of the organisms in his report on 41 patients, with consideration of specific serum therapy for pneumonia due to Bacillus Friedländeri. Avery,[5] Heidelberger and Goebel demonstrated chemical as well as immunologic relationships between Pneumococcus type II and B. Friedländeri of type B. According to Tomasek[8] (1925) and Quast[9] (1926) the rhinoscleroma bacillus can be distinguished by agglutination and complement fixation from Friedländer's bacillus and Bact. ozenae. It is possible that they form a special division of Friedländer group X. Differentiation is also made as to the form of the organism: "smooth" and "rough" strains are described. The smooth strains produce capsules, are type specific and virulent, the rough strains produce no capsule and no soluble specific substance and are not pathogenic.

The incidence of Friedländer's pneumonia is variously stated by different authors as from 0.5 per cent[10] to more than 5 per cent[11] of cases of pneumonia. Page[12] in 1934 reported the data collected on the bacteriology of acute infections of the middle ear and mastoid from 300 cases referred for myringotomy. He found the bacillus Friedländeri in two cases, or 0.7 per cent. I was impressed with his statement: "It is of some interest to note that there were two patients with infection due to Friedländer's bacillus group who recovered in a short time without operation on the mastoid." Later he states: "Almost three times as many operations on the mastoid were necessary in the cases in which the drums ruptured spontaneously as in those in which myringotomy was performed before rupture occurred." His article does not state whether in the two cases due to Friedländer's bacillus there was early myringotomy or not. It is probable that there was. In his group with infections due to pneumococcus type III, 35 patients were followed; 29 recovered without operation, and six, or 17.1 per cent, had operations on the mastoid. This is a lower percentage than given by other writers, notably Dean[13]. Kreutz and Witter[14] present the results of the study of 300 cases of acute mastoiditis in which simple mastoidectomy was performed. Cultures were taken from the mastoid cavities at the time of operation. Of 189 cases infected with hemolytic streptococcus eight died—approximately 4 per cent. Of 16 cases infected with pneumococcus type III, eight died, or 50 per cent. No Friedländer's bacillus was encountered in this series. Altmann[15], in 1935, reported seven cases of otomastoiditis caused by the Friedländer's bacillus. Mastoidectomy was performed in five of these cases and in two the lesion healed spontaneously. All the patients recovered. He remarks that the course of the disease is like that of a mucosus otitis, but the prognosis ap-

pears to be much better than that in infections caused by the type III pneumococcus. Kopetzky[16] states that this observation is contrary to what is found in the older literature, in which an infection with Friedländer's bacillus was regarded as one of great importance.

REPORT OF A CASE

Case 1. Man, aged 37, had a mild acute rhinitis and developed a fullness and tinnitus in his right ear. He had no pain and felt generally well. He thought he had impacted cerumen and consulted his doctor. He was informed that no cerumen was present and that inflations would give him relief. He had five Politzer inflations in the following ten days and was informed that his ear condition would clear up. He came to see me five days later. His chief complaint was a marked fullness in his right ear and the right side of his head and a dull earache; he had no mastoid pain nor tenderness. His temperature was 99.6 and white blood cell count 10,200. The ear drum was not bulging, but presented a dull grayish appearance. A myringotomy was performed under gas anesthesia. A platinum loop was used to obtain the specimen for the culture from the myringotomy incision. *B. mucosus capsulatus* was obtained in pure culture. It is of interest that at myringotomy the sensation felt was that of cutting through raw bacon; a sensation described by Ruttin which occurs when a myringotomy is performed in the presence of a streptococcus mucosus infection. Following the myringotomy the course was that of a mild subacute suppurative otitis media. The next day he had no pain, and though instructed to remain in bed, he made a short business trip. He had a moderate amount of thick drainage and less fullness than preceding the myringotomy. I explained to him the serious type of infection present in his ear and insisted that he remain at home, for undoubtedly this organism had been present since the onset of his fullness in the ear, sixteen days earlier. The next few days the temperature ranged from normal to 100.6 and the white blood cell count from 7,250 to 11,500; the fullness in the ear varied and he had an occasional pain. A week after the myringotomy, i. e., the 21st day since the onset of fullness, a slight mastoid tenderness was present on deep palpation. There was no sagging of the posterior superior canal wall but a moderate amount of thick drainage. He consented to a mastoidectomy only after persuasion and a positive x-ray report of mastoiditis. You will note that I mention the x-ray findings as a means of persuasion. Clinically he had a surgical mastoiditis, not because of the x-ray findings, but because of the time element indicated of a mucosus capsulatus otomastoiditis. Although it was only one week since the myringotomy, it was three weeks since the onset of the fullness in his ear. The mastoidectomy was interesting. The cortex of the mastoid was of moderate thickness, the trabeculæ of the cells beneath it were firm. The posterior canal wall was also firm. As the deeper walls were approached the trabeculæ were softer and in places destroyed. The amazing finding was the entire destruction of the plate covering the lateral sinus and the dural plate over the antrum and mastoid. A pure culture of *B. mucosus capsulatus* was obtained from the mastoid wound.

The post-operative recovery of the patient was uneventful. His ear drum and hearing were normal about a month after the operation, which was in January of 1937. Two weeks ago we made a culture from his tonsils on Loeffler's blood serum. A mixture of organisms grew including one colony of a sticky mucoid consistency, which on staining proved to be the gram-negative Friedlander's bascillus.

CONCLUSION

The *bacillus mucosus capsulatus* (Friedländer) rarely infects the middle ear and mastoid, and may present a clinical picture exactly like that resulting from the well known *streptococcus mucosus capsulatus*—the latter is known also as the *pneumococcus mucosus capsulatus* and *pneumococcus* type III.

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XXXIII

THE CLINICAL APPLICATION OF THE GALVANIC FALLING REACTION*

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Deviation of the body and head resulting from galvanic stimulation of the ear region has been reported in man and experimental animals by many investigators, but the clinical application of the galvanic falling reaction has been heretofore infrequently utilized, because of inconsistent results. Galvanic nystagmus has been much more frequently observed and reported than has the galvanic postural reaction, but the test of choice has usually been caloric and rotation stimulation of the labyrinth.

By 1874 as a result of the investigations of Augustine[1], Ritter[2], Purkinje[3], Hitzig[4], Breuer[5], and others, it had been demonstrated that closure of the galvanic circuit through the labyrinth produced anodal falling, and nystagmus toward the cathode, and that opening of the circuit caused a reversal of the direction of both nystagmus and falling.

Controversy arose as to whether the labyrinthine or retro-labyrinthine structures were stimulated by galvanic current. Investigations on frogs, fishes, guinea pigs, dogs, cats, and man were reported. The literature contains much in favor of each location. Many who failed to take in consideration the amount of current used, reported that similar galvanic reactions could be obtained in labyrinthectonized and normal animals.[6] An analysis of their results indicate that more current was required to obtain the same reactions in labyrinthectomized than in normal animals. Dohlman[7] considered Scarpa's ganglion as the location of stimulation by galvanic current. Jensen[8], Beyer and Lewandowsky[9], and Kny[10], individually demonstrated that experimental removal of the cerebellum failed to effect the galvanic reaction. Wilson and Pike[11] demonstrated that nystag-

*Read before the Chicago Laryngological and Otological Society, December 6, 1937.

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mus could be obtained by direct stimulation of the eighth nerve in animals. Neumann[12] obtained nystagmus in an exposed eighth nerve in the human. Ewald[13] was the first to demonstrate in pigeons that if a small amount of galvanic current is used, spread of current can be avoided, and the reactions thus obtained are entirely of labyrinthine origin. Later Jensen[9] with pigeons, Babinski[14] with pigeons, dogs, and guinea pigs, and Kollner and Hoffmann[15] with rabbits, and Blau[16] with cats, confirmed Ewald's findings.

James[17] in 1882 tested 43 students with a strong current, and reported nystagmus, vertigo, falling, stinging of the skin, subjective noises, flashes of light, and occasionally cerebral confusion on galvanic stimulation of the mastoid. He considered that central stimulation took place in his tests, and suggested that if only a small amount of current was used, the stimulation would be limited to the labyrinth. Pollak[18] in 1893 performed tests on 50 normals and 82 deaf mutes, and reported the characteristic falling nystagmus, and vertigo, with 5 to 13 milliamperes in those with labyrinths active to rotation. Babinski[14] noted anodal falling in normals. Alexander and Kreidl[19] reported falling toward the anodal side in a group of deaf mutes with active labyrinths. MacKenzie and Alexander[20] noted both galvanic falling and nystagmus, but recorded principally the latter.

In testing for galvanic falling some investigators had the patient stand upright with the feet together, or the heel of one foot in front of the toe of the other. Mygind[21] used the sitting position. Wodak and Fischer[22] had the patient stand upright with the feet together, the arms extended horizontally forward, and the eyes closed. Galvanic stimulation of the mastoid produced a slow falling reaction, which they termed the "discobolus reaction," because the position assumed resembled a discus thrower.

When I began studies with the galvanic falling reaction, I became confronted with the same problem many others investigating galvanic stimulation of the labyrinth had previously noted, namely the results were inconsistent.[23] The patient, due to apprehension, often maintained his equilibrium, so that no falling was present with 20 milliamperes or more, where the labyrinth was active, and the test had to be discontinued because of pain. Often a large amount of current was required to produce falling. The larger amounts of current produce pain, or a head turning due to skin stimulation, and may alter the reaction. Five milliamperes produce pain of varying degrees in normal individuals. The test as it was performed by previously described methods was abandoned because of inconstant results in normal individuals. The test to be of value must, (1) Abol-

ish voluntary interference by the patient, and, (2) Avoid large amounts of current which produced spread reactions, and pain, which besides being disagreeable produced movements in addition to those induced by vestibular stimulation.

To meet these requirements for a satisfactory galvanic falling reaction, the patient was placed in the normal standing position, eyes closed, on a platform, 16 inches wide, 21 inches long, and ¾ of an inch thick, under which was placed a fulcrum 3½ inches wide, and 1⅝ inches high. The edges of the fulcrum were beveled to permit less resistance to falling. The balance board was arranged with the fulcrum sufficiently wide so the patient could maintain his balance without effort when the circuit was open. Stimulation of an ear caused the patient, standing on the delicately balanced board, to fall to a side, since he could not voluntarily correct his displacement of the center of gravity caused by a change in muscle tone. There is a short latent period between the closure of the circuit, and the falling, which is not violent. Circular moist cloth covered electrodes are used, one over the mastoid, and the other on the upper portion of the sternum, permitting unilateral stimulation of the ear with small amounts of current, which I demonstrated in patients with one dead labyrinth. Galvanic current is painful to the auricles, and they must not be touched by electrodes, for the pain may complicate the interpretation of the falling reaction. The galvanic falling reaction obtained with the balance board is accurate, consistent, and easily recognized.

Two hundred and thirty-eight patients, 120 of whom were normals, were tested for the galvanic falling reaction with the use of the balance board. Closure of the galvanic circuit through the labyrinth usually with 1 to 2 milliamperes of current, rarely as much as 5 milliamperes, resulted in lowering of the platform toward the anodal side with each normal patient. There were no doubtful reactions. The falling was present without visible nystagmus (convex lenses were not used), when the eyes were permitted to be open for this observation. Vertigo was absent, or occasionally only slightly present. Patients described the sensation as one of being pushed or pulled over.

One hundred and eighteen pathological subjects, patients of Doctors Lewis J. Pollock and Loyal Davis, and myself, were tested for galvanic falling, and included 63 patients with epilepsy, 43 idiopathic, and 20 patients, organic type, all active to the caloric test, six with mastoidectomies with cochlea defects, and one with a bilateral suppurative otitis media, all active to rotation and caloric stimu-

lation, and patients with one of the following conditions: acute otitis media, otitis externa, myasthenia gravis, migraine, trigeminal neuralgia, a recovered polio-encephalitis, a labyrinthine fistula, and a recently recovered serous labyrinthritis were examined for their galvanic falling reaction. All of these 78 patients with pathological conditions fell toward the anode on closure of the galvanic circuit with 5 milliamperes or less current.

A group of 21 patients with intracranial neoplasms, verified at operation by Loyal Davis, included tumors at the following locations: Five, cerebellar; four, fronto-temporal; one, frontal; three, parietal; two, brain stem; two, neuromas of the eighth nerve; one, tentorial tumor partially involving the eighth nerve; one, post-operative cerebello-pontile angle tumor involving the eighth nerve, and one, temporal meningeoma. Fifteen of these patients, including five with cerebellar neoplasms; four with fronto-temporal; one, frontal; three, parietal, and two, brainstem neoplasms, all active to caloric stimulation, fell toward the anode on galvanic stimulation of the ear while standing on the balance board. Five additional patients with suspected intracranial neoplasms were active to caloric stimulation and galvanic falling. Four of the group of patients with verified intracranial neoplasms, included two with neuroma of the eighth nerve, one with a postoperative cerebello-pontile angle tumor, and one patient with a dead labyrinth opposite the side of a temporal lobe tumor, failed to respond to caloric stimulation, or to fall with stimulation of 15 milliamperes on the involved side. One patient who proved to have a left tentorial tumor involving the eighth nerve, failed to react to caloric stimulation of the left side, but fell toward the anodal left side with 8 milliamperes of current. This patient's right ear reacted normally to caloric stimulation, and galvanic falling. One patient with a right temporal memingeoma failed to react to caloric stimulation of the right labyrinth, but fell toward the anode with 5 milliamperes of galvanic current. The left labyrinth reacted normally to caloric and galvanic stimulation. Patients with supra-tentorial neoplasms required as a rule less current than the infra-tentorial group. Patients with hyperactive labyrinths, requiring only .5 milliamperes of current included those with the following conditions: A labyrinthine fistula, a suspected intra-cranial lesion, a verified parietal tumor, and a recently recovered serous labyrinthritis. Eleven pathological subjects with inactive labyrinths to caloric stimulation included the following conditions: Two patients with chronic suppurative otitis media with cochlear deafness, three patients with old basilar skull fractures, one patient with Ménière's syndrome, and six patients with cochlear deafness which followed an infectuous disease or whose

cause was unknown. These failed to fall with 15-20 milliamperes of galvanic current.

To confirm our impression that galvanic falling performed with a small amount of current was due to peripheral and not brain stem stimulation, Loyal Davis and I conducted experiments on cats which was reported at the 1936 A. M. A. convention in connection with a clinical report[24]. We decerebrated two cats by the anaemic method described by Pollock and Davis[25]. The experiments of Magnus and De Kleyn[26], Pollock and Davis[27], and others demonstrated that the decerebrated cat is an excellent experimental animal with which to test labyrinthine postural reactions, because the complicating and inhibitory effects of the higher centers are released. A cat decerebrated above the nucleus of the seventh nerve was placed in the position for maximum extensor tonus, namely, on his back with the nose 45 degrees above the horizontal. Neck reflexes were controlled by fixing the cat's head to the examining table. One electrode was clamped in the ventral mid-line of the neck. The second electrode was inserted in the auditory bulb in the roof of the mouth. The electrodes were connected to the same galvanic machine that we had used to test galvanic falling clinically. On closure of the galvanic circuit with from 1 to 2 milliamperes extensor tonus diminished in the forelimb on the anodal side, and simultaneously the extensor tonus in the opposite forelimb increased. Repeated tests with the electrodes in either auditory bulb revealed the same phenomenon of antagonistic increase and reduction in the extensor tonus of the forelimbs. This experiment is analogous to our clinical test in that the balance board immediately registers the change of tonus in the limbs of the patient whose ear is stimulated with galvanic current. Our decerebrated animal failed to show nystagmus on stimulation of the ears with galvanic current, with ice water or rotation, because the pathways concerned with nystagmus were destroyed. Another cat had both eighth nerves sectioned at the exit of the internal meatus. Ten days later this cat was decerebrated by the anemic method. This decerebrated cat with both eighth nerves sectioned was subjected to the same galvanic tests that were conducted on the first animal. With 20 milliamperes of current, no change in extensor tonus occurred on closure of the circuit. When stronger current was used it spread throughout the animal.

CONCLUSION

1. The galvanic falling reaction is an accurate simple clinical test for the vestibular postural reaction as demonstrated by our examination on 237 patients.

2. Experimental evidence obtained on decerebrated cats indicate that the galvanic current as we used it with the balance board to produce falling is not localized to the brain stem, but has a peripheral action.

3. Since it is almost generally agreed by present day physiologists that the otolithic apparatus is concerned with posture, further investigations may prove that the galvanic falling reaction is a test for otolithic function, and that a large series of examinations may lead us to isolated lesions in the vestibular postural pathway.

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XXXIV

CONGENITAL ANTERIOR HERNIATIONS OF BRAIN*

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Maldevelopments of the skull during fetal life are not infrequent, and when they occur, protrusion of portions of the brain through the membranous anlage of the skull frequently results. Such protrusions are usually noted at the sites of the fontanelles, i. e., occipital, frontal, sphenoidal or mastoid, and less frequently through the cribriform plate of the ethmoid, the foramen cecum, an enlarged foramen magnum, or through one of the various suture lines. The majority of these herniations are cystic, and are composed of thinned-out cerebral cortex enclosing a cavity which communicates with the ventricular system, and therefore contains cerebrospinal fluid. According to Browder,[1] similar tumors are found which are solid, and still more rarely they are not connected to the brain by a demonstrable pedicle. These, Berger[2] believes, are true new growths.

It is the purpose of this paper to review briefly the recorded history of the condition, to describe its pathogenesis, and to present a case report of one example of the condition which has recently been under my care.

In a rather careful, though by no means exhaustive review of the literature, I have been able to find some forty cases of anterior encephaloceles and have seen references to some thirty or thirty-five cases in the older literature which apparently resembled the more detailed ones. The occipital hernias are more frequent, but are not included in this series. The anterior hernias are usually divided into basal and sincipital varieties, and in reported cases the two occur with about equal frequency, though both Meyer[3] and Hopkins[4] have made the observation that probably the basal types are more frequent than reports would seem to indicate, because of the fact that the intranasal growths very frequently resemble ordinary nasal polyps and are so regarded by the examining physician. I shall limit a brief description of cases to the sincipital varieties, of which there are eighteen cases in this series.

*Read before South Texas District Medical Society, April 22, 1938.

The fundamental difference between the two types is that the sincipital types always present themselves as external tumors—on the nose, orbital margin or forehead—while the basal varieties may be intranasal, intrapharyngeal or intraorbital. The growths have been variously designated as fibroglioma (Rocher and Anglade),[5] glioma (Schmidt),[6] (Clark),[7] (Sussengrath),[8] encephalocele or hydrencephalocele (Schotz),[9] (Guthrie and Dott),[10] (Natansen),[11] (Malek),[12] encephaloma (Berger),[2] rhinencephalocele (Browder and de Veer),[13] and, by the majority of authors, merely hernias of the brain.

Berger,[2] in 1890, discussed the pathology of the condition and drew the distinction between cystic growths which he considered to be hernias, and solid ones, which he thought were true new growths.

In 1882, Heinecke[14] presented his classification of basal encephaloceles, and in 1890, von Myer[3] gave his classification of the sincipital varieties. Both are on anatomical grounds, and there are three types of each. Of the sincipital type we have:

(1) Encephalocele naso-frontalis, where the pedicle passes through a defect at the junction of the nasal and frontal bones. The tumor will be visible externally in the midline at the base of the nose.

(2) Encephalocele naso-ethmoidalis, where the defect lies between the frontal, nasal and ethmoid bones, and the tumors appear at the junction of the cartilaginous and bony parts of the nose.

(3) Encephalocele spheno-maxillary, in which the pedicle passes through the superior or inferior orbital fissure into the sphenomaxillary fossa. Of this type, Fenger[15] says: "The tumor can be felt in the mouth on the medial side of the ascending ramus of the inferior maxilla, and is visible on the outside of the cheek below the zygoma." Of the three types, the naso-frontal is much more frequent.

PATHOGENESIS

Any discussion of the origin of these benign tumors involves a review of the anatomy and embryology of the skull, since it seems probable that whether they are merely extrusions of brain substance or are true new growths, their original exit from the skull is made possible because of clefts which are present either normally or pathologically between the various bones during their development. The pathogenesis of the basal and occipital tumors varies from that of the sincipital varieties only because of location, but a brief survey of the

development of the bones involved in sincipital hernias will serve to illustrate the process of growth of the tumors elsewhere.

The frontal bone arises from a right and left center of ossification, each appearing at about the end of the second month of fetal development. Several other centers appear before birth and fuse with the main center. The two centers approach each other, but unite only after birth—about the eighth year. Failure of these to approximate normally might result in an extrusion of the brain in the midline of the forehead at the junction of one or the other half with its respective nasal bone, resulting in a naso-frontal encephalocele.

The ethmoid bone develops from several centers of ossification in the cartilaginous nasal capsule, which surrounds the nasal cavity above and laterally. About the end of the fifth month, a nucleus appears on each side in the lateral wall, from which the cribriform plate develops and from which the turbinates ossify. At the end of the first year a nucleus appears in the upper part of the cartilaginous nasal septum and from this the crista galli and lamina perpendicularis ossify. One case (Browder and de Veer),[13] must have developed prior to this time, for the crista galli was absent. In the sixth year, the two lateral and middle parts of the ethmoid bone unite with each other and with accessory centers which appear in the crista galli and lamina cribosa after birth. Ossification is not complete until the sixteenth year.

The nasal bones develop from membranous bone formed in the anterior part of the cartilaginous nasal capsule from a nucleus which first appears at the end of the second month.

The lacrimal bones are membranous, arising from a nucleus on the outer surface of the cartilaginous nasal septum in the third month.

Any maldevelopment of these structures might result in defects between frontal, nasal, ethmoid or lacrimal bones, with consequent encephalocele formation. The naso-orbital variety protrude through the point of union of the lacrimal, frontal and nasal bones, while the naso-ethmoidal type denotes a defect between the frontal, nasal and ethmoid bones.[16]

More than one hundred years ago, Geoffrey St. Hillaire[17] offered the sound explanation regarding the origin and development of these growths which, with further explanations, is the accepted theory today. In 1827 he published his adhesive theory, and attributed the

growths to adhesions (caused by intra-uterine pressure) between the dura, brain and skin of the cranium, which resulted in an arrest of the development of the bony vault and a subsequent outgrowth of the brain.

As is customary in medical circles, his contemporaries promptly arose to attempt a refutation of this theory, and in 1829, Himly,[18] and in 1831, Serres,[19] insisted that the extrusion took place through clefts which are normally present during ossification rather than abnormal slits which follow maldevelopment.

Spring,[20] in 1854, attempted to clarify the question, and after considerable investigation came to the conclusion that since so many of the tumors were connected with the ventricles, they probably arose because of a hydrocephalic enlargement, and he explained all of them on the basis of dilatation of the anterior horns of the lateral ventricles (frontal tumors), third ventricle (trans-sphenoidal tumors) or fourth ventricle (occipital tumors). He explained the rarer solid growths by supposing that the hydrops existed only for a short time and reduced itself later during intra-uterine life, leaving only the solid tumor with no trace of a connective ventricular passage into the growth.

Klementowsky,[21] in 1868, offered his belief that these growths were due to cranio-tabes, rickets, or syphilis, with the resulting malformation of the skull; but his position was shown to be untenable because only a small percentage of cases reported showed any evidence of these conditions.

Ankermann[22] believed the solid tumors to be the result of a generalized microcranial development within which the normally developing brain could not be contained. On the other hand, he explained the cystic types as being due to hydrocephalic changes, thus corroborating Spring's theory, in part, at least.

By 1890, Myer[3] came to the conclusion that St. Hillaire's theory was the most satisfactory one for the various types of tumors, and he presented a case of solid tumor which he believed could not be explained on the basis of the then generally accepted theory of Spring.

Berger,[2] in 1890, also accepted St. Hillaire's theory for most cases, but he believed the solid growths to be true new growths, because of the wide variation in microscopic structure of the tumor and of tissues at the supposed site of origin. To these he gave the name encephaloma, and he reported two cases.

In 1937, Sternberg[23] again reviewed the development of the growths and explained it on the basis of embryological facts with which St. Hillaire was probably not familiar.

Sternberg[23] points out that as the neural groove in the young embryo closes, the anterior and posterior ends close last, and remain open for about a week after the rest of the tube is closed. These are the anterior and posterior neuropores which normally remain open until the end of the third week. The anterior neuropore corresponds to the foramen cecum of the frontal bone. In young embryos this spot lies on the forehead at the upper rim of the area triangularis; in the more mature embryos it lies at the level of the root of the nose, between the eyes. As stated previously, this neuropore normally closes at the end of the third week and is separated from the skin by the ingrowth from each side of mesoderm, which will later form the skull. If, however, the connection persists, mesodermal invasion is impossible and the skull development will be faulty at this point. Only a thin epithelial membrane will then separate the brain from the surface and herniation of a greater or less degree will result. Usually, in such cases, mesodermal invasion is arrested entirely, and the brain and tumor remain connected by a pedicle. Occasionally, however, the mesodermal invasion is only retarded long enough to permit herniation of the brain and then proceeds to such an extent that at term there will be a complete severance of the herniated mass from the brain from which it arose. Even in these cases, however, the bony defect usually persists.

Sternberg[23] reports one case in which there was, in addition to an anterior encephalocele, an absence of the corpus callosum, and since this structure originates from the region of the anterior neuropore, he believes that in many other cases of anterior encephalocele the corpus callosum would be found wanting. He is unable to find, however, any mention of abnormality of the corpus callosum in reported cases of encephalocele. Conversely, in reported cases of absence of the corpus callosum, no mention of anterior brain herniations are mentioned. It seems inconceivable to me that either condition would have been overlooked in the course of pathological study of the other. Nevertheless, Sternberg believes there is a close relation between the two abnormalities.

At any rate, thanks to Sternberg, we now have what seems to be a completely adequate explanation on embryological grounds of the adhesive theory of St. Hillaire, which he offered a hundred years ago on what appears to have been a purely empirical basis.

Olson,[24] in discussing the etiology of these growths, quotes Haggard as giving the following possible factors in addition to the more generally accepted theory which we have discussed above:

(1) Union of brain sac with amnion, interfering with development of bone.

(2) Disease of bone, causing nonunion or porosities, with protrusion of brain covering through the aperture, probably as existing with disease of the brain itself.

(3) Intra-uterine hydrocephalus with bulging as the bones come together.

(4) Exostoses or protuberances on the pelvis of the mother, upon which the fetal head rests, interfering with development.

REVIEW OF PREVIOUS CASES

In addition to the case reported by Sternberg, there have been some seventeen cases of sincipital tumors reviewed for this discussion. Bruns[25] reported a case in 1860 of a naso-frontal tumor in a child who was perhaps five years old. No details of his management of the case are given. Volkmann[26] reports a naso-frontal encephalocele in a baby eighteen months old. Birnar[27] reports a case in a 30-year-old woman who came to his attention at the time of her accidental death. No antecedent history was available. Autopsy revealed a naso-frontal encephalocele. In addition, she has a complete bifid nose.

Of the remaining cases reviewed, ten were naso-frontal, four were naso-ethmoidal, one was naso-lacrimal. My own case is of the naso-ethmoidal variety.

Mittendorff[28] removed a growth from the naso-ethmoidal region in a four-day-old infant. Recovery resulted.

Hildebrand's[29] case was a large naso-frontal tumor in a fourteen-day-old baby. Attempted removal resulted in death.

Three cases of naso-frontal type are reported by Grynfeldt,[30] Herigoyen[31], and Binet and Mutel.[32]

Hopkins[4] reports a case in an eleven-months-old baby. There was an external tumor on the right side of the nose and also an intranasal polypoid tumor. The intranasal growth was removed, the external portion being left undisturbed. Death resulted from meningitis. He makes the interesting comment: "This case is presented as a curiosity rather than because such lesions present practical possibil-

ities of relief at our hands . . . if the protrusion is into the nasal cavities, the idea of operation should not be entertained."

Clark's[7] case was one of two reported by him, the other being of the basal type. His case was in a two-year-old male with a tumor in the midline at the naso-frontal junction. Removal was followed by recovery. No pathological study was made and no mention was made as to whether it was cystic or the rarer solid type which Berger believes are new growths.

Nordlund[33] and Miller[34] report cases, naso-frontal in type, where successful removal was accomplished.

Funkhousen[35] reports successful removal of a tumor which traversed the foramen cecum of the frontal bone where a one and one-half-inch defect was encountered. The patient was seven weeks old.

Stuart's[36] case was that of a new-born infant. The tumor was quite large and increased in size when the baby cried. There were no other abnormalities. The bony defect was at the naso-frontal junction. Operation was performed and the child lived for some three weeks, but succumbed as a result of general debility.

Begler[37] reports a fronto-ethmoidal tumor in a fourteen-months-old baby, and there was an associated microphthalmia. Operation was advised and refused.

Browder[1] reports a case of an eleven-months-old baby with a tumor attached by a pedicle to each frontal lobe; the pedicle leaving the cranial cavity through an opening at the foramen cecum. The tumor was removed but death resulted in five days from pneumonia.

Browder and de Veer[13], in an excellent review of the subject, present a case in which the tumor arose from the rhinencephalon, and although other authors do not mention this source of origin, these writers believe that many of them probably do arise from the rhinencephalon. From their pathological studies they conclude that there is considerable basis for support of Berger's contention that the wide difference between the structure of the tumor and the brain tissue at or near the point of origin justifies the supposition that at least the solid types are true new growths.

Olson[24] reports a case of a child two and one-half months of age with a fronto-ethmoidal tumor. Operation for removal was followed with uneventful recovery.

There are several factors which seem to me to point against the theory that these tumors are true new growths. In the first

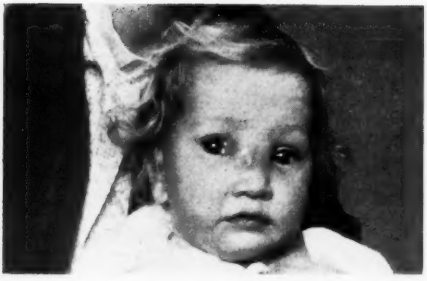

Fig. 1. I. L. P., age 2, showing naso-ethmoidal encephalocele.

place, their location is suggestive of their origin as a result of embryologic malformations, for they are always either at the fontanelles, suture lines or junctions of various bones or of their component ossification centers. In the second place, although the tissue of the tumors differs somewhat from that of normal brain tissue, still there is definite differentiation; even in the solid tumors, into brain and glial cells, and the arachnoidal and dural coverings are easily recognizable as such. These can frequently be shown in serial sections to be actually continuous with the coverings of the brain within the skull. Thirdly, there is the fact that the majority of these cases are cystic and show a direct connection with the ventricles.

REPORT OF A CASE

Case 1.—I. L. P., age 2, Italian female, was brought in because of a tumor of the right side of the nose, close to the inner canthus of the right eye; and was referred to me by their ophthalmologist, whom they had consulted first. (Fig. 1). The child was a normal full term baby, having had a normal delivery, growth and development. She had been perfectly healthy, with no previous illnesses or abnormalities. The mother stated that the growth had been present since birth and was relatively no larger now than it was at birth. There was no noticeable change in size or position by crying or change of posture.

Physical examination was essentially negative except for the tumor. This was firmly attached, could not be reduced by pressure, and had a firm cystic feel. It was well walled off and lay just beneath the skin on the right side of the nose. It was the size of a small marble. The bridge of the nose was wider than normal —a point which earlier writers call attention to as being characteristic of this type of tumor.

No bony defect could be palpated. There was no abnormality within the nose, and nasal breathing was in no way impaired. Laboratory findings were negative.

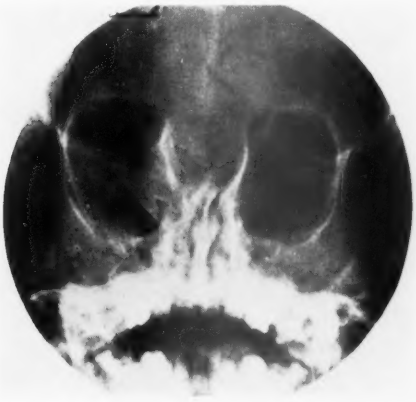

Fig. 2. Anterior view, showing distortion of nasal bones and resorption of floor of frontal sinus.

X-ray studies (Fig. 2) revealed a beginning development of the fronto-ethmoidal cells, a defect in the floor of the frontal bone, and a similar defect of the right nasal bone. Other bony development was apparently normal, including the crista galli and the sella turcica. The roentgenologist was of the opinion that we were dealing with a mucocele of the right ethmoid labyrinth.

Operation was decided upon. An incision one-half inch long was made and the dense capsule of the tumor was encountered almost immediately. The tumor shelled out easily over its lateral and anterior surfaces, but it became at once apparent that we were dealing with a solid tumor which had extended through or had eroded the frontal and nasal bones. The capsule was apparently continuous with the dura, for it could not be separated without going through it. This was done and the tumor was removed in its entirety. There was no pedicle or other connection between the tumor and the brain and the dural edges were free from internal adhesions. Flow of cerebrospinal fluid was free. The wound was closed with dermal sutures. Postoperative course was marked by a profuse drainage of cerebrospinal fluid for eighteen days, but this gradually diminished, and on the 26th day recovery was complete. Patient was seen four months after operation and was entirely well. There was considerable contraction of the scar, so that the inner canthus of the eye was pulled downward, interfering somewhat with lacrimal drainage. This, I believe, was due to the fact that the nasal bone and a portion of the frontal bone either never developed or were eroded away by the tumor, depriving the soft tissues of proper support. It is probable that a plastic operation will be necessary at a later date in order to avoid or correct a saddle nose.

Ethmoid mucocele could not be definitely ruled out pre-operatively. X-ray suggested it as the most likely diagnosis. However, the presence of the tumor since birth and the entirely negative history of nasal troubles of any sort ruled against this possibility.

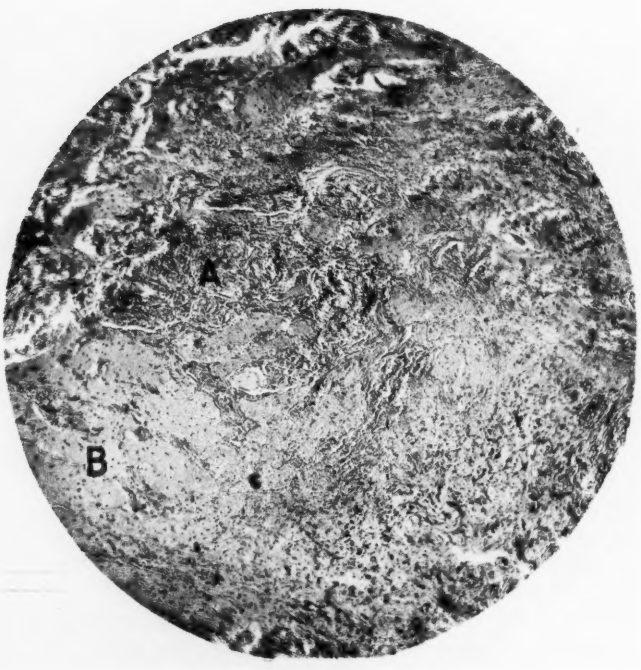

Fig. 3. Microphotograph of tumor section. *A*. Stroma. *B*. Glial tissue.

The tumor (Fig. 3) was sent for pathological study and the report is as follows: "Frozen section made through center of tumor shows dense fibrous tissue in irregular strands, some more cellular fibrous tissue, and a small amount of fat on the surface. Here also are some small bundles of striat muscle. Scattered through the tumor are areas of tissue hard to identify in frozen section. These were thought at first to resemble cell masses in dural endothelioma, allowance being made for the embryonal type of tissue. On more careful study I believe that much of this material is nervous or glial. Careful celloidin preparations confirmed this latter opinion. There is no evidence of malignancy." Diagnosis: Benign herniation of brain tissue.

CONCLUSION

(1) A review of anterior cerebral herniations with a summary of theories of pathogenesis is presented.

(2) A brief review of some twenty reported cases is given.

(3) A case of a two-year-old girl who was successfully treated for a naso-ethmoidal hernia is reported.

1304 WALKER AVENUE.

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XXXV

SINUS THROMBOSIS

PART II

LONGITUDINAL SINUS THROMBOSIS

CULLEN WARD IRISH, M.D.

LOS ANGELES

Only occasionally do spastic paraplegias with bladder symptoms, due to softenings in both paracentral lobules or leg motor areas, appear in involvement of the superior longitudinal sinus, no focal symptoms usually presenting. The scalp is edematous and its veins are engorged. Convulsions are frequent and signs of increased intracranial pressure, due to hydrocephalus, develop early. The difficulty in diagnosing the condition is exemplified by four cases reported by Doyle[75]. In the absence of meningitis, the early onset of apathy or stupor in a patient with evidence of thrombosis of the transverse sinus secondary to suppurative otitis media generally indicates infectious thrombosis of the superior longitudinal sinus by retrograde extension, especially if associated with choked disks and convulsions, he points out. In but five cases of all supratentorial operations performed at the Mayo Clinic in nearly 16 years was there occlusion of the sinus, all by endotheliomas. When the probable presence of tumor or of inflammatory disease can be excluded, Jacksonian seizures, showing progression from one foot to the other, or beginning in the foot and gradually involving the homolateral upper extremity, suggest impairment of the circulation of the cerebral veins and probable thrombosis of the superior longitudinal sinus. Abrupt onset of symptoms of increased intracranial pressure, which follow a fluctuating course, should suggest the possibility of superior longitudinal sinus thrombosis as well as ventricular tumor. The absence of the element of progression after a fair length of time, or actual regression of symptoms, is suggestive of thrombosis of the superior longitudinal sinus. If the syndrome is associated with edema of the eyelids and dilation of the veins of the eyelids and forehead, and if fracture of the skull, orbital and periorbital infections and thrombosis of the cavernous sinus can be excluded, the diagnosis is established, according to Doyle's dissertation. Hassin[76] has reported a case

of complete obliteration of the longitudinal and both transverse sinuses without internal or external hydrocephalus. He concludes that such a condition has no effect on the absorption of the spinal fluid from the subarachnoid space.

SUPERIOR LONGITUDINAL SINUS THROMBOSIS

Thrombosis of the superior longitudinal sinus occurred in 16 instances, 18.0% of all sinus thromboses and 3.1% of all thrombotic processes in this survey. The patients were all Caucasians except one, a negro. Ten were males and six females. The age incidence was two months to two years in six, one each was 5, 6, 13, 25, 37, 38, 62 and 72, and two were each 53 years old, averaging 20.6 years. Their hospital residence was from five hours to 25 days, averaging eight days, except in the case of an amaurotic family idiot, 65 days, in whom the time of the thrombotic formation could not be definitely established. The duration of the illness in the remainder, from onset of the symptoms of the thrombosis till death was from six hours to 33 days, averaging 13 days.

The origin of the condition in four of the nine children was otitis media. In a 10-months-old babe, pyelitis and bilateral otitis media were the sources. Acute upper respiratory tract infections preceded the middle ear infections except in the one just noted and was present in another who developed no ear infection. A supraorbital abscess, spreading periostally over the entire forehead, was the source of the complete longitudinal sinus thrombosis in a 13-year-old boy (S. T. 1). Hand twitching, neck rigidity and convulsions marked the onset in a two-year-old child with vegetative endocarditis as the infectious source of the thrombosis. Diarrhea two weeks previously was followed by a red, spotty, exanthema before tremors on the right side and stupor indicated cerebral involvement in an 18-months-old baby. Amaurotic family idiocy had been diagnosed in a 14-months-old child four months previously and the symptoms of this disease masked the signs of onset of the sinus thrombosis found at necropsy.

In the adults, the onset of the cerebral involvement was shown by hemiplegia in three, by stupor and coma in four, subsequent to respiratory infections in two of them and following paranasal sinus infections in two patients.

The superior longitudinal sinus and the veins emptying into it were thrombosed in seven cases. The lateral sinuses were also involved in three and the petrosal sinus with them in another. The superior longitudinal, left cavernous and inferior petrosal sinuses were thrombosed in one instance. Both transverse sinuses were also occluded in

one specimen with the thrombotic processes extending down into the jugular veins. Marked subcortical softening had occurred in three brains and hemorrhage was also found with the superior longitudinal sinus thrombosis, in four patients,—a massive subpial and subarachnoid hemorrhage over the left cerebral cortex in one, hemorrhage over the left cerebral cortex in one, hemorrhage and softening in the right posterior parietal and occipital region in one, diffuse subarachnoid hemorrhage in another and subpial and petechial hemorrhage in the fourth. Meningitis coexisted in three specimens.

Brief summaries of the histories of the illnesses in some of the children demonstrate the progress and symptoms shown, frequently with little diagnostic evidence of the cerebral involvement.

S. T. 7. The history given regarding the illness in a five-year-old girl was that she had suffered an attack of measles several months before and bilateral otitis media with spontaneous rupture of both membrani tympani one month previously, neck pain and enlarged cervical glands having been present since. Acute coryza with fever had been present a week when she was brought to the hospital. She was uncooperative and resistive and presented positive bilateral Kernig reactions and neck rigidity. The white blood count was 12,500 with 82% polymorphonuclear leucocytes. The spinal fluid was under 16 mgm. of mercury pressure, showed increased globulin, 1150 cells, 73% polymorphonuclear leucocytes and 27% lymphocytes, and normal sugar content. Meningococci were found in the smear and 30 ccm. of antimeningococcic serum were administered after withdrawal of 35 ccm. of cloudy fluid. The next day 15 ccm. of serum was given intraspinously and an equal amount intramuscularly, with marked clinical improvement the following day after 15 ccm. more serum was administered intraspinously. Five days later a retropharyngeal abscess was incised. At this time the eyelids were puffy, and twitching of the facial muscles was observed. Death occurred two days later. The superior longitudinal and both right and left transverse sinuses were found to be filled with partially adherent thrombi extending into the jugular veins. Two of the larger cerebral veins entering the superior longitudinal sinus were also occluded. No exudate was present. Coronal sections disclosed the presence of small, petechial hemorrhagic areas throughout the white and gray substance. The right mastoid was soft and necrotic and an adjacent pachymeningitis was present from which the thrombotic material had originated.

S. T. 11. A male baby was losing weight and developed inanition fever of 103 degrees when 13 days old. Two days later bilateral

myringotomy was done, but no drainage ensued. The second day afterward, streptococci were found in a smear from the ears. The babe became progressively weaker and died on its twenty-fifth day. His mother was in a tuberculosis ward. At necropsy thrombosis of the superior longitudinal and right lateral sinuses was disclosed. Bilateral otitis media was present with the right middle ear containing thin, purulent, yellowish-brown exudate. Some bone necrosis had developed and a subdural abscess had extended into the wall of the right lateral sinus. The upper portion of the right cerebral hemisphere was somewhat softened by minute hemorrhages into its substance.

S. T. 8. A 14-months-old male was admitted to the hospital after a week's cough and vomiting with fever as high as 103 degrees F. for three days. Bronchopneumonia was found, and a week later, three days before death, the left ear was discharging.

Complete thrombosis of the entire longitudinal and right lateral sinuses was found present. The numerous veins entering the superior longitudinal sinus were also thrombosed and masses of dilated veins and hemorrhage were shown over the left frontoparietal and right frontal and temporal areas. Bilateral otitis media existed, but there was no evidence of direct extension of infection through the bone to the lateral sinuses.

S. T. 6. Persistent cough from a pertussis infection had been present for five months in a 6½-months-old male baby, when he was admitted to the hospital, acutely ill, with rhythmic jerking of the spastic left arm and leg. The eyelids were swollen and a positive Kernig reaction was elicited on the right side. Bilateral myringotomy resulted in profuse drainage. The white blood count was 23,100, polymorphonuclear leucocytes being 63% and large lymphocytes 37%. The spinal fluid was under increased pressure and the cell count decreased from 762 cells to 10 per ccm. in five examinations in eight days. Tests for the presence of tubercle bacilli were negative. Smears showed no organisms and no culture growth was obtained. Cultures from the ears gave growths of streptococci. The colloidal benzoin curve was 000,002,333,331,000. Following much improvement, stupor appeared and increased, the left arm and leg became progressively weaker, the head and eyes were held to the left and death occurred the twenty-first day in hospital.

A suppurative thrombosis of the entire superior longitudinal and lateral sinuses ending at the jugular foramina was shown. Also several of the cerebral veins entering the longitudinal sinus were

thrombosed and a mass of dilated veins with subarachnoid hemorrhage entirely covered the right occipital, parietal and postfrontal lobes. No gross meningitis was present.

S. T. 3. A female two years of age. The right hand began to twitch a week before she was brought to the hospital. On admission she had general convulsions every two to three minutes, which could be produced by touching the child anywhere. The fixed pupils were dilated and the jaws tightly set. Some neck rigidity was present. The slightly cloudy yellowish spinal fluid was under some increased pressure and contained 35 cells per ccm. The superior longitudinal sinus, the superior cerebral and superior cerebellar veins were distended with thrombi. The other sinuses and jugular vein were clear, but the inferior sagittal, the great vein of Galen, and both internal cerebral veins were thrombosed. No exudate was present and the accessory sinuses and mastoids were negative. An acute vegetative endocarditis was present. Cultures from the vegetations and the throat gave growths of hemolytic streptococcus.

S. T. 4. A history of fever and diarrhea for four days, two weeks before hospital admission was given in the case of a boy of 18 months. Red spots scattered over his face and body were noted for a week before "trembling" of the right side occurred and he was brought to the hospital stuporous and presenting frequent general convulsions. Bilateral Babinski reactions were elicited. Discharge of whitish, mucoid material followed a left myringotomy. Later, both middle ears drained. The white blood count was 16,650, polymorphonuclear leucocytes 58% and lymphocytes 42%. The spinal fluid was negative. Albumen and casts were present in the urine. Smears from the ear discharge showed staphylococcus aureus. The blood Wassermann reaction was negative. Death occurred the seventh day after the onset of signs of cerebral involvement shown by the abnormal motor activity. The brain showed an organized thrombus and greenish yellow purulent material filling the superior longitudinal sinus and extending back nearly to the torcular herophili. The cerebral veins draining into the superior longitudinal sinus were all thrombosed and multiple small hemorrhages had occurred in the adjacent cortex. The sphenoid sinuses contained pus.

In adults suffering sinus thrombosis, the symptoms present are often perplexing and intracranial localization difficult, frequently due to other coexisting thrombotic or hemorrhagic processes and meningeal involvement. Three patients in this group of seven developed hemiplegia as one of the prodromal signs of cerebral involvement.

S. T. 16. A 25-year-old negro suffered sudden right hemiplegia and motor aphasia following exercise the morning of his hospital admission. A history of some recent antiluetic treatment was obtained. Examination disclosed unequal, irregular fixed pupils, right hemiplegia including the face and motor asphasia, and urinary incontinence. Deep reflexes on the right were exaggerated. His pulse was 84, temperature 98.2 degrees and respiration 20, and blood pressure 145/90. He was conscious, but restless and somewhat mentally confused and clouded. Repeated lumbar punctures disclosed increased cerebrospinal fluid pressure as high as 400 mm. of water and bloody fluid, later zanthrochromic. The blood Wassermann and Kahn reactions were reported 4 plus and the spinal fluid negative. The clinical temperature increased to 103.6 degrees and he died the ninth day after the appearance of the sudden hemiplegia. A large thrombus was found in the superior longitudinal sinus. The dorsolateral surface of the left cerebral hemisphere was covered with subarachnoid and subpial hemorrhage from the superior left cerebral veins. Coronal sections showed a massive hemorrhage in the left cerebrum filling the left ventricle.

A male 72 (S. T. 9) had a history of an upper respiratory infection, varying in severity, for a number of weeks. For a month there had been some fever and periods of mental confusion and clouding of consciousness progressively increasing. Seven days before admission to the hospital, right hemiplegia had appeared and the next day motor aphasia increasingly severe. He was admitted in coma which persisted the three days he survived. The longitudinal, left cavernous and inferior petrosal sinuses were found thrombosed and a basal meningitis with the exudate extending up the Sylvian fissure was present.

Occasional severe headaches had been present for seven years in a female aged 37 (S. T. 12), admitted to the hospital in coma. Five years previously she had been under observation in the psychopathic department where meningovascular lues was diagnosed. She had received consistent intramuscular and intravernous antiluetic treatment since until an influenzal attack four months ago. Four days before entering the hospital, projectile vomiting had occurred and three days later sudden right hemiplegia appeared. The next day she had a generalized convulsion and subsequently was irrational until coma developed 18 hours later. Examination disclosed fixed pupils, right hemiplegia with exaggerated reflexes on the right side, temperature of 102.6 degrees F., and pulse of 120. A previous spinal fluid Wassermann reaction had been reported positive. Death occurred the

eighth day of her hospital residence. The entire brain was found to be greatly softened and mushy. Thrombosis of the longitudinal sinus extended into the meningeal vessels. Coronal sections showed extreme softening just beneath the cortex extending through both frontal and parietal lobes. The cerebellum and medulla were uninvolved.

Many sinus infections were reported by a male aged 53 (T. 240), who was admitted to the hospital with a spastic paralysis of the right upper extremity. He had a severe coryza and profuse discharge from the left nostril which had been present for two weeks. The right extremity reflexes were exaggerated and the neck was stiff. Reports of the blood examinations included a normal red cell count, white cells 37,600, polymorphonuclear leucocytes 92% and a negative Wassermann reaction. The spinal fluid pressure was 20 mg. of mercury. The fluid was clear and contained 76 polymorphonuclear leucocytes per cmm. Smears showed no organisms and no culture growth was obtained. The colloidal benzoin reaction showed a meningitic curve. The left frontal and ethmoid sinuses were exposed the day after his hospital admission and he died three days later. At necropsy a septic thrombotic process filled the superior longitudinal sinus and left superior cerebral vein. The paranasal sinuses were clean, the middle ears and sphenoid sinus negative.

A female of 62 (S. T. 14), who was admitted to the hospital comatose, soaked with perspiration, survived but a short time following the onset of symptoms. She had been ill three days with a cough and fever and had fainted the day before she was brought to the hospital, where she lived but five hours. No localizing signs of cerebral pathology were elicited. The pupils were equal and regular, and deep reflexes equally diminished on the two sides. The blood pressure was 120/90. Examination of the brain disclosed diffuse recent subpial hemorrhage, 6 cm. wide, over the superior and lateral surface of the right cerebral hemisphere beginning at the pre-central gyrus and extending back to within 3 cm. of the posterior extremity of the occiput. The superior longitudinal sinus, the veins emptying into it from the right cerebrum and the right lateral sinus were filled with non-septic thrombotic processes. A few cerebral veins of the left cortex were thrombosed. A 3 cm. area of reddish-gray softening was present in the left superior and lateral aspect of the right occiput involving the white matter also. The subpial hemorrhage had distended the right cerebral sulci as much as 2.5 mm. and some cortical softening had resulted.

S. T. 10. A 10-months-old female babe was brought to the hospital because of fever, pain on urination, foul smelling urine and

SUPERIOR SINUS THROMBOSIS

Series No.	Autopsy No.	P. F. No.	Initials	Color	Sex	Age	Involvement Other Than Superior Sinus
1.	218	132-684	M. P.	White	M.	13	Supraorbital abscess
2.	548	151-923	R. S.	White	M.	14 mo.	
3.	1027	177-983	A. P.	White	F.	2	Cerebral and cerebellar veins
4.	6404	98-728	M. C.	White	M.	1½	Cerebral veins
5.	6438	100-364	A. E.	White	F.	53	Cerebral veins
6.	7139	134-007	R. L.	White	M.	6 mo.	Bilateral lateral sinuses and cerebral veins
7.	7150	135-930	M. d'A.	White	F.	5	Bilat. transverse sinus and bilat. jugular veins
8.	7164	129-799	R. M.	White	M.	14 mo.	Right lateral sinus and right lateral veins
9.	7305	117-806	K. McA.	White	M.	72	Inf. petrosal and Right cavernous sinuses
10.	7426	147-480	H. H.	White	F.	10 mo.	Right middle cerebral cortical embolus
11.	7518	152-212	B. D.	White	M.	25 da.	Right lateral sinus
12.	8214	192-232	M. H.	White	F.	37	Meningeal veins
13.	8331	124-101	M. O.	White	M.	2	Cerebral veins
14.	8942	230-050	L. B.	White	F.	62	Right lateral sinus cerebral veins
15.	9723	266-429	R. R.	White	M.	53	Superior cerebral veins
16.	10182	287-348	S. W.	Black	M.	25	Massive left subarachnoid hemorrhage

tenderness in the kidney region. The urine had a specific gravity of 1012 and contained much albumen and many pus cells. The white blood cell count was 22,000 with 96% polymorphonuclears. The day after admission a left myringotomy resulted in a moderate sanguineous discharge. Four days later, the temperature suddenly rose to 105 degrees F. rectally. A bilateral myringotomy was done with resulting sanguineous discharge from both middle ears. Following treatment for the pyelitis present the urine was negative for organisms the ninth day of hospitalization, but at this time neck rigidity appeared and lumbar puncture showed the spinal fluid to be bloody. The child became progressively worse and ten days later a bilateral mastoidectomy was performed. Free pus was found in the right mastoid antrum with periantral necrosis present. The spinal fluid smears and culture were negative. Cultures from the ear gave a growth of staphylococcus aureus. Death occurred the fourth postoperative day, 24 days after hospital admission.

The brain was found to be soft and edematous. There was a 1.5 inch area of hemorrhage and softening in the right posterior parietal and occipital region to which a posterior cortical branch of the right middle cerebral artery was traceable. Extending from this area into the superior longitudinal sinus was an antemortem thrombus completely filling it. Pyelonephritis was present on the left side and thrombosis of the left renal vein and the inferior vena cava. Septicemia was the final cause of death.

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PART III

CAVERNOUS SINUS THROMBOSIS

CULLEN WARD IRISH, M.D.

LOS ANGELES

The first account of cavernous sinus thrombosis was given by Abercrombie[77] in 1818 in his descriptions from autopsy findings, and in 1821 Duncan[78] also observed the condition postmortem. Vigla[79] made clinical reports in 1839, followed by DeCastelnau and Ducrest[80] in 1846. The differential diagnosis was considered by Knapp[82] in 1868. Hartley and Knapp[85] were the first to attack the sinus surgically.

The infrequency of the condition is indicated by ensuing reports.

Dwight and Germain[87] in 1902 collected 178 cases from the literature and reported four cases, including one cranial operation. D. Smith,[104] in reporting five cases in 1918, estimated less than 300 cases had been recorded. Chisolm and Watkins[105] found but eight cases in 50,000 surgical records of Johns Hopkins Hospital in 1920.

The literature suggests the diagnosis is rarely made in the absence of exophthalmos and ocular chemosis, although the latter is common. Many cases must be overlooked, indicating the need for careful search for cavernous sinus thrombosis in the presence of septicemia and pyemia.

The three most common causes are marasmus, trauma and infection. Marantic thrombosis usually occurs in association with debilitating conditions at the extremes of life, the cavernous sinus then always being involved secondarily to thrombosis of the longitudinal or lateral sinuses. The traumatic form is usually associated with fractures of the base of the skull, more rarely from direct injury to the sinus through the orbit or by accidental injury during operations on the Gasserian ganglion. Septic thrombosis is by far the most common form. It is nearly always secondary to thrombophlebitis of some of the afferent or efferent veins. It may be primary in the cavernous sinus, due to direct extension from a basilar meningitis, abscess of the postorbital space or suppuration in the sphenoid sinus or petrous portion of the temporal bone.

The symptoms recited by Chisolm and Watkins[105] are: (1) Those due to venous obstruction, namely, exophthalmos, edema of the retina and of the eyelids and the bridge of the nose: (2) Those due to involvement of the neighboring cranial nerves, the chief symptoms being ptosis, dilation of the pupil, oculomotor palsies, loss of the vision, pain in the ophthalmic distribution of the fifth nerve and very rarely trismus from irritation of the motor branch accompanying the mandibular division of the fifth nerve: (3) Symtoms due to general sepsis, fever, chills, sweats, rapid, small and thready pulse. Often there are symptoms of meningitis, of pulmonary embolism and of embolism elsewhere. Differential diagnosis needs to be made from erysipelas, cellulitis of the orbit, tumors of the orbit and arteriovenous fistula of the internal carotid artery.

Eagleton[118] describes acute and chronic types. In the acute type the symptoms are those of sudden obliteration of a large venous trunk from an acute and complete blockage of the veins emptying into the cavernous sinus. In the chronic form no symptoms of venous engorgement appear because a slow occlusion of the sinus has allowed time for the venous blood to establish other channels.

Acute infection of the cavernous sinus results from extension of a thrombophlebitis of the veins which empty into the sinus anteriorly—the ophthalmics—associated with a sudden occlusion of the lumen of the sinus. The classical signs of chemosis and exophthalmos are caused by the purulent thrombophlebitis reaching the sinus by way of the ophthalmic veins. The infection is in the eyelids or ethmoid cells, traveling through the pterygoid plexus from the teeth or by the facial and angular veins from the lip or orifice of the nose. This acute obliterating form of septic thrombophlebitis is most common, not only on account of the proximity of the inflammation, but also because all the venous radicles of this region have few valves, which not only allows a retrograde thrombophlebitis but, by permitting a reversal of venous current, is favorable to the formation of a thrombus, with this resulting "ebb and flow" movement of the blood. The process is early accompanied by chemosis and exophthalmos, first of one eye then the other, associated with profound pyemia and followed by death from sepsis or an accompanying complicating meningitis.

Slowly obliterating thrombophlebitis may occur without localizing signs, as the production of exophthalmos and chemosis requires a sudden disturbance of the return venous circulation before the collateral circulation has time for readjustment.

A classification of the condition is also presented by Eagleton[118] on the basis of the route of invasion into the sinus.

(1) In the ophthalmic type (anterior), the infection enters the anterior part of the cavernous sinus from in front by way of the ophthalmic vein. Following an infection, generally staphylococcic, of the lip, eyelid, nasal orifice, teeth or the anterior part of the mouth, there develops a retrograde thrombophlebitis which ascends through the angular or facial venous plexus, enters the orbit and is transmitted by the ophthalmic veins to the cavernous sinus. This is also the usual route of invasion of the ethmoid cases that perforate the orbit.

(2) In the pterygoid type (anterior inferior), the infection attacks from its forward and lower region. In this instance the infection reaches the sinus from below by way of the pterygoid plexus as it ascends in the pterygoid fossa with its numerous anastomoses from the pharyngeal plexus, through the foramen ovale, the deep facial and inferior ophthalmic veins. These infections usually originate from foci in the posterior teeth, the jaw or maxillary antrum and less frequently in the pharynx or tonsils. When the cavernous sinus thrombosis originates from the tonsil, the path of invasion is generally indirect, involving first the internal jugular vein and from it ascending into the inferior petrosal sinus, but there are records of direct extension from the tonsil into the pterygoid plexus in which the tonsillar veins anastomose.

(3) The sphenoidal or mesial infection attacks the mesial portion of the sinus by caries of the sphenoid or by a thrombosis of veins which pass through the sphenoidal roof into the cavernous sinus.

(4) The aural (posterior) infection enters the posterior portion of the sinus from thrombosis of the inferior petrosal sinus or from caries of the deep cells at the apex of the petrous pyramid where it impinges on the body of the sphenoid and unites with the petrous portion of the opposite side.

(5) The tonsillar or posterior-inferior course is through the pterygomaxillary fossa by way of an ascending thrombophlebitis of the jugular vein. The sinus is attacked first in the lower and posterior portion by way of the petrosal sinus.

(6) A microscopic pathway occurs in the direct infection into the lumen of the cavernous sinus from the carotid venous plexus. Here a jugular bulb infection from either the ear or throat extends through the internal carotid canal in the skull and thus reaches the

anterior portion of the cavernous sinus. Thrombophlebitis of the venous plexus surrounding the internal carotid artery stands clinically between the aural or posterior and ophthalmic anterior types, as, although of aural origin, it early gives rise to exophthalmos.

(7) Mixed types. All six types, although anatomically and clinically distinct, may pass from one to another because of the free anastomotic communication of the venous radicles with each other.

Cavernous sinus thrombosis from staphylococcic infections frequently causes valvular vegetations, as demonstrated by Whitehead.[90] Dislodgement of septic emboli frequently occur with common terminal manifestations, giving rise to infective infarcts in the kidneys, spleen and lungs and multiple small abscesses in the liver, frequently the cause of jaundice.

The diagnostic criteria are: (1) A known site of infection: (2) Blood stream infection: (3) Early but slight symptoms of obstruction, such as (a) a temporary fullness of the veins of the retina, (b) the Crowe-Beck sign of filling of the retinal veins on compression of the carotid artery and jugular veins on the affected side and (c) temporary ocular edema from continued pressure on the neck: (4) Neighborhood symptoms from involvement of the nerves in the sinus, by inflammatory edema or direct pressure, causing paralysis or paresis of the third, fourth and sixth nerves or paresthesia or anesthesia in the first branch of the fifth nerve, manifested by transient or permanent diplopia, herpes of the face and lips and pain in the teeth or supra- or intra-orbital regions or behind the eye: (5) Neighborhood abscess in the adjacent soft parts secondary to a retrograde septic thrombophlebitis extending from the cavernous sinus into the smaller venous radicles which normally empty their blood into the cavernous sinus or its immediate accessories. By this method of extension abscess may result in the orbit, occiput, neck or nasopharynx. (6) Symptoms of complicating diseases, e. g., headache and papilledema from serous meningitis of the basal cisterna. When there is venous stasis within the ophthalmic vein exophthalmos and chemosis result.

Repeated blood cultures are indicated as one positive culture is diagnostic in a suspected blood stream infection. The leucocyte count is significant and should be made daily. In septic cavernous sinus thrombosis it will vary from twenty-one to forty and as high as fifty-two thousand.

The prognosis is bad, Dwight and Germain[87] in 1902, in their study of the 178 reported cases finding only a seven per cent recov-

ery rate with expectant treatment. Chisolm and Watkins[105] in 1920 found that of those operated upon less than seven per cent had recovered.

Eagleton states that surgery is helpful if instituted early, and meningitis is either protective or confined to the basal cisterna or subdural space contiguous to the sinus. Ligation of the carotid artery gives rest to the parts, particularly the cavernous sinus. Tying the internal carotid artery similarly supplies rest but does not control surface hemorrhage so well and takes longer to perform. He has rarely seen untoward effects of ligation of the common carotid artery except in three cases, in all of which other factors entered.

Chisolm and Watkins believe the location of the focus of infection should be the determining factor in the route of approach. If the focus is in the orbit, the orbital route of Mosher might be employed, if in the ethmoids or the sphenoid sinus, the ethmoidal-sphenoidal route of Langworthy would be indicated. The surgical failures are evidently due to the early occurrence of meningitis and thrombosis of neighboring sinuses.

Reverdin[83] reported ten observations of purulent phlebitis of the facial vein starting at the level of the commissure of the lips and extending up to the inner canthus of the eye, but stopping there or continuing via the transverse vein of the face to the external jugular vein and from there by the anastomosing branch to the internal jugular vein. Death occurred from involvement of the cavernous sinus and concomitant intracranial lesions or by a portion of the clot, containing multitudinous organisms, breaking off and being carried by the venous circulation to the right heart and thence to the lungs.

Lenhartz[88] gave the postmortem findings in eight cases of staphylococci infections following furuncles of the lips, nose and eyelid. There is a striking tendency for infectious thrombophlebitis of the facial vein to follow lip infections. There are no valves in the facial vein which lies in firm muscular and fibrous tissue containing little connective or fatty tissue, but the labial veins have numerous valves, according to Poirer and Charpy.[89] Lenhartz[88] was opposed to early incisions and believed the first principle of treatment was rest with the avoidance of trauma.

Powers[86] advised early and wide incision of lip furuncles following his knowledge of the death of a young physician two days after the onset of a furuncle on the upper lip. Lanz[106] does not believe in incision of lip furuncles, after death following the incision by an

expert of an upper lip furuncle in a fellow interne. Ludlow[81] considered these infections of the lips and face as malignant pustules, but Thomas Smith[84] entirely dissented from this. Walters[94] treated a case of severe facial carbuncle without incision, with recovery. Keppler[97] reported twelve cases of facial infection, only four of which were severe, treated by Bier's constriction hyperemia method without incision. Lexer[100] reported fatal sepsis following a single treatment of a small lip furuncle by suction. Both Lexer and Wrede[96] advised wide incision through indurated areas when a progressive suppurative phlebitis was present. Bradburn,[103] in writing on the abortive treatment of boils, pointed out the danger of attempting to abort them by puncture or the injection of phenol into the indurated tissue.

Walton Martin,[111] in reporting eight patients with extensive lip infections, of whom six died, believed early operation or manipulation tends to spread and not limit the infection. He admonished against ever using a local anesthetic. If the abscess is localized or intolerable pain is present he advises the use of a sharp knife. If it is necessary to incise widely, the incision should follow the line of incision for resection of the superior maxillary. Usually no local treatment is of avail when the infection is widespread and the sinus thrombosed, according to Dwight and Germain.[87] Gallemaerts[115] advocated every furuncle of the face should be treated early and radically by galvanocautery. Tavernier[95] reported incision of the cavernous sinus, one patient living a few hours afterward and the other a few minutes.

The jugular and facial veins have been ligated in the effort to limit the infection. Lexer[100] reported a carbuncle of the cheek with purulent thrombophlebitis of the facial vein and general infection, in which he avoided further metastasis by ligation of the jugular vein, with recovery. Ligation of the jugular vein should be considered in the presence of a blood stream infection and signs of general sepsis, according to Martin, though usually lung lesions are present when the patient is first seen. Tavernier[95] mentions ligation of the jugular vein by Lexer, but notes it is very difficult to recognize thrombosed veins in the plexus and numerous anastomoses. Obviously, ligation should only be done on a sound vein wall at an appreciable distance from the infected thrombus. There is much danger of disturbing or loosening the thrombus in the endeavor to ligate, as the nasal or labial vein branching from the point the ophthalmic enters the angular vein is only about one inch long. Many simple circumscribed staphylococcic infections are changed into

widespread dangerous lesions by ill-advised treatment at the onset of the infection.

CAVERNOUS, PETROSAL AND SIGMOID SINUS THROMBOSIS

These sinuses were thrombosed, usually bilaterally or multiply, in 34 patients in this survey, 38.6 per cent of all sinus thromboses and 7.57 per cent of all thrombotic processes in this survey. These patients were all Caucasians except two negroes. Twenty-two were males and twelve females. Their age incidence varied from two months to 62 years, averaging 28.4 years. Those under 10 years of age were, 2 months, 18 months, 5 and 8 years; three were 11 and 12; two 14 and one each 18 and 19 years old. Seven were 22 to 27; three were 33 to 37; eight were 40 to 48; three 51 to 58 and one 62 years of age. Their hospital residence with this condition was from one hour to 35 days, averaging 5.1 days and the survival periods varied from 24 hours to 35 days, averaging 7.75 days.

The infectious sources of these 34 instances of sinus thromboses were comedones and small furuncles in and about the nose and upper lip in 14, the infection beginning in the upper lip in four, in the anterior nares in six, from the forehead in one, in the cheek in two and from a small furuncle just below the ear in another. Otitis media was the etiological source in four, the frontal sinus in one (S. T. 70) and the sphenoid sinuses in another (S. T. 67). The sinus thrombosis occurred in the presence of toxemia of pregnancy in an 18-year-old primipara (S. T. 57) and followed swimming in a 14-year-old boy (S. T. 68), followed a tonsillectomy in a female of 52 (S. T. 67) and dental extractions in two (S. T. 71 and 85). The sinus thrombosis occurred in the presence of measles with otitis media in a boy of 8 (S. T. 82), followed gangrenous diphtheritic pharyngeal slough in a 5-year-old girl (S. T. 59), and originated from rhinitis in two (S. T. 64 and 87), the latter with otitis media later developing. Erysipelas was the infectious source in one (S. T. 84), extension of pneumococcic meningitis the cause in two and the cause not determined in two.

Meningitis was present in 13 of the 34 patients and an extradural (S. T. 79) and small temporal lobe abscesses had formed in four, and an abscess under the temporal muscle in one. A subdural clot was present over the right cerebrum in the two-months-old babe (S. T. 80).

The left cavernous sinus alone was thrombosed in five instances, the right in six. The cavernous sinuses were bilaterally thrombosed in ten and involved bilaterally with other thrombotic processes, including the ophthalmic veins in two of them, in six other cases.

Headache, fever and varying impairment of consciousness, usually with irritability, delirium or coma were the common prodromal symptoms of the sinus thrombosis. Facial cellulitis was present where the infectious source was about the lips or nose. Bilateral exophthalmus was present in four patients and unilateral in two others. Fundal changes were noted in four and various oculomotor involvements in five. Neck rigidity was marked in six patients, Kernig reactions in four. Spasticity of the extremities appeared in five. Two patients, a boy of 11 (S. T. 66) and a 22-year-old female presented hemiparesis and one (S. T. 76) nystagmus and right facial weakness with profuse exudate being found over the cerebral hemisphere and particularly the right temporal lobe and in the right sylvian fissure.

A number of case abstracts are arranged as to the type of sinus involvement, in the order delineated in the catalogue of the patients in this group.

The following brief histories are examples of five instances in which the left cavernous sinus was found involved.

S. T. 55. Constant headache and painful stiff neck following sore throat had been suffered by a white female of 40 for two days when she was admitted to the hospital. There was a furuncle on the forehead and a recent scar in the left cheek. The left eyelids were swollen, partially closing the eye. Kernig reactions were positive bilaterally. The leucocyte count was 10,950 with 90 per cent polymorphonuclears. The urine had a specific gravity of 1,030 and contained 1-plus albumen and 3-plus acetone. The spinal fluid was turbid and contained 12,400 leucocytes per ccm., but no organisms were isolated from its smears or culture. Two days later the spinal fluid was cloudy and contained 457 cells, 90 per cent leucocytes per ccm. Thirty cc. of antimeningitic serum was administered. The neck was rigid and the left eyelids and forehead skin were reddened and swollen with the development of facial erysipelas. Blood and spinal fluid Wassermann reactions were reported negative. The colloidal benzoin spinal fluid curve was 000,022,000,122,222. Death occurred the third day of hospital residence.

The brain was edematous and congested. Yellow, purulent basal meningitis was found. The left cavernous sinus was thrombosed, the path of infection being along the veins between the inner canthus of the left eye and the cavernous sinus. The lungs contained numerous purulent infarcts, smears from which were pure staphylococcus aureus. An acute splenic tumor was present. Smears from the sinus contained staphylococci.

S. T. 56. A white female of 22 was admitted to the hospital with swollen, purple, induration of the entire left neck and face, entirely closing the eye. A pimple had appeared in the left angle of of the mouth ten days previously, had been opened a few days later and reopened several times since. The swelling had persisted and increased to involve the entire left face and neck. The leucocyte count was 15,000 with 80 per cent polymorphonuclears. The urine had a specific gravity of 1,022 and contained albumen 4-plus and granular and cellular casts. Blood cultures gave a growth of staphylococcus aureus. She died within 4½ hours of her admission.

The left cavernous sinus exuded 2 cc. of thick sango-purulent material. The right cavernous sinus was only slightly reddened. The lungs contained numerous small white, purulent areas and red pneumonic consolidation.

S. T. 57. A left cavernous sinus thrombosis was the only cerebral pathology present in an 18-year-old female, pregnant and near term, who entered the hospital on account of headache, vertigo, vomiting and edema increasing the previous few days. Her temperature was 99 degrees, pulse 90 and blood pressure 114/100 on admission. The urine contained (2-3 plus) albumen, occasional casts and specific gravity as high as 1,025 at several examinations. The red blood cell count was 3,750,000 and hemoglobin 60 per cent. The blood sugar was 47 and nonprotein nitrogen 21 gms. per 100 cc., the carbon dioxide combining power 48.2 volume per cent. Sixteen days later labor began and was completed in three and three-fourths hours by extraction with episiotomy. Shock was evidenced at the completion of the delivery. The red blood count was 2,900,000; hemoglobin 50 per cent. She became cyanotic and comatose the next day and died during a blood transfusion.

The left cavernous sinus was found to contain pus. The paranasal sinuses were normal. The death certificate was signed "Toxemia of Pregnancy," contributory, "Shock—Postoperative."

S. T. 58. Headache, nausea and vomiting, chills and fever had been present one week when a white male of 26 was admitted to the hospital. His temperature was 105.8 F., pulse 130 and respirations 32. He was irrational and jaundiced. There was present a slight proptosis of the left eye, paralysis of the left external rectus, protrusion of the tongue to the left and stiffness of the neck. A systolic apical murmur was auscultated. The spinal fluid pressure was 400 mm. of water; it was cloudy and blood tinged and contained 800 leucocytes per ccm. The sugar was greatly decreased

and the globulin content increased. Cultures gave a growth of staphylococcus albus. Death occurred 14 hours after his admission.

A moderate subarachnoid hemorrhage was sufficient in amount to give the brain surface a pink discoloration. There was no gross purulence except in a 1 cm. area on the anterior margin of the left cerebellar hemisphere overlying the jugular foramen where it was slightly adherent. The ventricles were filled with fluid blood. Yellow pus, under pressure in the left cavernous sinus, had extended from the necrotic purulent condition of the cavernous dura around into the dura of the posterior intertransverse sinus and basilar plexus. The left carotid sheath was purulent and necrotic in appearance and there was necrotic and purulent material in the regions of the left cavernous sinus and the left internal jugular foramen. The middle ear and right mastoid were absolutely clean, the left mastoid contained a thin film of bloody, serous fluid, microscopically but not grossly purulent. The left external jugular vein presented a very severe purulent phlebitis. The lining of the vein showed numerous subendothelial abscesses. The right internal jugular vein was normal. There was a 15 mm. area of osteomyelitis of the skull around the left jugular foramen. A septic, gray splenic tumor was present. The liver showed cloudy swelling with icterus and the lungs multiple septic infarcts. An acute mastoiditis was present on the left.

S. T. 59. A 5-year-old female idiot with congenital lues, who had been undergoing antileutic treatment under institutional supervision for a year, was admitted to the communicable disease department with diphtheria of three days duration. She had received 20,000 units of antidiphtheritic serum. Toxin-antitoxin had been administered eight months previously. When admitted the temperature was 102 degrees, pulse 120 and respirations 24. The extremities were spastic, the wrists flail and no mental responses were obtained. Numerous throat cultures were positive for diphtheria and 20,000 more units of antidiphtheritic serum were administered. The left cheek became red and swollen but little other change was shown the 21 days she survived.

The dura at the base in the middle fossa, particularly on the left side, showed grayish-green discoloration and thickening. The left sphenoid bone under this area showed advanced osteomyelitis which also involved the basilar and central portion of this bone. The left cavernous sinus contained the same type of fluid necrotic material which was present over the sphenoid bone. There was superficial slight discoloration over an inflamed area in the left temporal lobe. A foul gangrene of the pharyngeal tissues extended

unbroken to the area of osteomyelitis in the floor of the left middle fossa. There were multiple septic infarcts and gangrenous abscesses in the right lung.

The following brief histories are examples of four instances in which the right cavernous sinus was found involved.

S. T. 61. A 14-year-old girl picked a papule in front of the right ear a few days before she was admitted to the hospital. The right side of the face was red, hard and indurated. The lesion had spread into the neck and upward, covering the entire right side of the head and neck. The temperature was 103 degrees, the pulse 100, respirations 18. The leucocyte count was 11,950 with 78 per cent polymorphonuclears. Several blood transfusions were given but death occurred the sixth day.

Pus was present below the right temporal muscle. The right cavernous sinus was thrombosed. There was staphylococcic cellulitis of the right face. Both lungs contained multiple infarcts and there was empyema of the right pleura.

S. T. 62. Both cavernous and the right lateral sinuses were involved in a boy of 14 who was brought to the hospital in coma with a history of fever, vomiting, and swelling about the right eye for two days. The day previous to the onset of symptoms he had been well when he went swimming, but the next morning the eye was swollen and painful. The spinal fluid was cloudy and showed a pleocytosis. Pus discharged from under the right middle turbinate and bilateral ethmoid, hence antral windows were made. A blood culture positive for staphylococci was obtained. Death ensued two days later, four days after the eye swelling first appeared. A moderate basilar meningitis and yellow pus at the tip of the right temporal lobe with slight hemorrhagic softening was found at examination of the brain. The fluid in the lateral ventricles was turbid. The right lateral sinus contained a purulent clot, the right cavernous sinus a number of small thrombi and pus, but no thrombus was present in the left cavernous sinus. There was no pus in the auxiliary sinus though the ethmoid was edematous and hyperemic. The lungs contained a number of scattered infarcts.

S. T. 63. The right cavernous sinus was found thrombosed in a male 51, who five days before admission to the hospital had scratched a small furuncle in the right nasal meatus. The second day after he "felt badly" and had a severe headache. The next day he was nauseated, vomited and seemed feverish. The nasal furuncle was lanced. He was admitted to the hospital semicomatose showing

edema, swelling, redness and cyanosis of both orbital regions. Both eyeballs protruded and there was some drainage from the right nostril. Some general spasticity was present throughout and all deep reflexes were exaggerated. The blood pressure was 170/100. He lived but seven hours after he was brought to the hospital. An early basilar meningitis on the under surface of the right temporal lobe to past the midline was found at autopsy as well as the right cavernous sinus thrombosis. In the lungs, 1 mm. to 1 cm. gray areas were shown under the pleura which on section were seen to be small abscesses surrounded by areas of pneumonia. Smears from the meninges and lung abscesses showed staphylococci.

S. T. 64. A 22-year-old male had a coryza for three days, profuse nasal discharge for two days and headache the day before he was admitted to the hospital in coma. There had been epistaxis the day of entry. The temperature registered 103 degrees axillary, pulse 84 and respirations 30. The neck was rigid and positive Kernig reactions were elicited. The blood cultures gave a growth of nonhemolytic streptococcus. The spinal fluid was under 275 mm. of water pressure and contained 1,000 cells, 96 per cent leucocytes per ccm. and 3-plus globulin. Death occurred the second day.

There was yellow purulent exudate over the frontal lobes and at the base, most particularly about the cerebellum. The cortical veins were intensely hyperemic. The right cavernous sinus contained pus and an intrasinus abscess. Both mastoids contained much dark, orange-red sticky serum. Both middle ears contained pus, the left twice as much as the right. There was frank pus about the nerves in the right internal meatus extending along the facial nerves to the right middle ear cavity. Smears from the meninges showed the presence of streptococci.

The following brief histories are examples of nine instances in which bilateral cavernous sinuses were found involved.

S. T. 66. An 11-year-old boy who had been in a boys' camp for two weeks and recently had ridden his bicycle into a parked car, possibly injuring his head, had been sick for two days with frontal headache, pain behind his ears and fever, when brought to the hospital with a diagnosis of poliomyelitis. The temperature registered 104 degrees, the pulse 100, blood pressure 112/48. The neck was slightly rigid. The white blood cell count was 11,500 with 78 per cent polymorphonuclears. The spinal fluid contained 36 lymphocytes per ccm. Poliomyelitis and antimeningitis sera were administered. Four days later it contained 850 and 1,000 polymorphonu-

clear leucocytes per ccm. Bilateral positive Queckenstedt responses were obtained. The boy was irrational at this time. Three days later a flaccid left hemiplegia appeared and a right subtemporal decompression was done with a postoperative diagnosis of basilar meningitis being established. He died fifteen days after he was admitted to the hospital.

There was a slight amount of yellowish purulent exudate over the base of the brain, mostly about the optic chiasma and laterally into the sylvian fissures. The sphenoid and ethmoid sinuses contained a large amount of purulent exudate under slight tension. The bony plate superior to the right sphenoid and to the posterior ethmoid sinuses was eroded and partially necrotic. An extradural abscess extended from here along the greater wing of each sphenoid bone and down into the middle fossa. The pituitary was floating in purulent exudate. Each cavernous sinus contained septic thrombi which extended into the circular and the superior portions of the superior and inferior petrosal sinuses on each side. That portion of the ophthalmic vein nearest each cavernous sinus was thrombosed on each side. The brain was negative as was also the skull for fracture. Smears from the exudate contained staphylococci.

S. T. 67. Ten days following a tonsillectomy, a white female of 52 was found unconscious in her bed with the head retracted and the legs flexed. She was admitted to the hospital in coma, showing subconjunctival edema with slight hemorrhage and bilateral exophthalmus. The pulse rate was 136 and blood pressure 176/46. Bilateral Kernig reactions were elicited. The leucocyte count was 9,600 with 93 per cent polymorphonuclears. The spinal fluid contained 1,900 cells per ccm. and an increase in globulin content, but no organisms were isolated from its smears or culture. Death occurred within 18 hours of her admission to the hospital.

The brain demonstrated the increased intracranial pressure by the flattened convolutions and the marked congestion of the vessels. At the base was a localized collection of thick exudate. On the floor of the skull, within the sella and about it was a large amount of greenish-black, foul smelling purulent material beneath the dura extending on either side about the cavernous sinuses which were both completely filled. The left ophthalmic vein was thrombosed similarly and the retro-orbital fat was edematous and gangrenous about equally on the two sides. There was some necrosis of the clinoid processes. The mucous membrane of the ethmoid cells was edematous and numerous polyps, some as long as 2 cm., were within both the anterior and posterior ethmoid and also the sphenoid cells.

There was some dark, purulent material about the internal carotid arteries but their lumina were patent. The lateral sinuses and middle ears were negative.

S. T. 68. Osteomyelitis of the left mandible was the source of the sinus thromboses in a white male of 40. There had been much pain in the left lower jaw for five days, for which three left lower molars had been removed two weeks before he was brought to the hospital. Following the dental extractions the jaw had become increasingly swollen and painful. Roentgenological examinations of the jaw showed no osteomyelitis but the dental sockets were not healed. The blood Wassermann reaction was reported positive (4+). An attempt to drain the mandibular abscess resulted in no drainage. The second day after admittance the neck was rigid, the pupils were contracted and fixed and subconjunctival edema appeared. The leucocyte count was 59,600 with 85 per cent polymorphonuclears. The urine and spinal fluid were negative. Death occurred the second day after his admission to the hospital.

Necropsy examination showed (1) absence of the left lower molars, with sloughing and a gangrenous socket about the first molar cavity; (2) a gangrenous opening in the floor of the mouth near this socket extending into an extensive abscess beneath the left jaw which involved the left parotid gland and the tissues on the mesial aspect of the ramus of the left mandible; (3) septic thrombophlebitis of both cavernous sinuses and purulent sphenoid sinusitis with local extension involving the pituitary gland and attaching itself to the inferior aspect of the left temporal lobe near the midline, with the formation of a 5 mm. abscess in the temporal lobe cortex; (4) the left maxillary sinus was markedly purulent. The other sinuses and the middle ears were negative. The brain was edematous and the meninges generally congested and inflamed. There were occasional small areas of degeneration of the thalami. Smears from the sinuses contained streptococci.

S. T. 69. Six days before entering the hospital a 27-year-old negro had a furuncle in the right nostril opened by his physician. Swelling of the upper lip and left eyelids had steadily increased since, and four days later chills and pain in the left chest appeared and had increased in severity. When admitted he answered questions with much difficulty. The left eyeball was reddened, its lids swollen and the conjunctiva edematous and injected. The upper lip was edematous and the submaxillary region indurated. The temperature registered 104 degrees. Incisions were made in the upper lip and a rubber dam drain inserted. There was a slight drainage

into the mouth from the nasolabial area. Death occurred the second day after his hospital admission.

Both cavernous sinuses contained septic thromboses, the venous meshwork being filled in with sanguineous exudate. The cerebral convolutions were flattened but there was no meningitis. Coronal sections showed edema and moderate congestion with occasional minute petechial hemorrhages in the white matter. There were multiple septic infarcts in the lungs and an acute splenic tumor. Smears from the sinus thrombi contained staphylococci.

S. T. 70. A history of frontal sinusitis two years previously, followed by tonsillectomy and adenectomy, with only occasional attacks since was given by a white male of 19 when he was admitted to the hospital. The present attack had begun four days ago and the day of his admission fever and stiff neck had appeared. No pathological reflexes were elicited. The leucocyte count was 22,200 with 92 per cent polymorphonuclears. The spinal fluid was under increased pressure and contained 2,250 cells, 60 per cent leucocytes per ccm. Improvement occurred until two days later, when the temperature increased to 103.4 degrees and the pulse rate to 154. The spinal fluid contained 4,450 cells, 80 per cent leucocytes per ccm., but smears and cultures were negative. The frontal sinuses were treated by the Lathrop method with insertion of a rubber drainage tube. The right frontal sinus contained a pyogenic membrane and the left was completely filled with pus under pressure. Death occurred the third day after hospital admission. The frontal sinuses were partially filled with bloody fluid but no free pus. The sphenoid sinus was filled by 10 cc. of foul smelling, viscid, greenish-brown, purulent material. The middle ears and the mastoids were normal. Both cavernous sinuses were filled by pus similar to that in the sphenoid sinus.

S. T. 71. A white female, aged 62, died twelve hours after her hospital admission, sought because of cellulitis of the left face following a tooth abscess. She had always been in good health until three weeks before when a coryza of several days duration was followed by a toothache in the upper left jaw. The left face, including the left eyelids, swelled and one week later the right eyelids became edematous. At this time the tooth abscess ruptured into the mouth and the facial swelling subsided somewhat, but severe headaches persisted. Chills and fever were present for a few days. When examined on admission she had been unable to see with the left eye for two weeks, the entire left face was greatly swollen and the left eye protruded. Purpuric spots were scattered over the chest and

abdomen. No neurological localizing signs were present and the reflexes were normal. Urinalysis showed albumen 2-plus, granular and hyaline casts were present.

At necropsy green purulent material filled both cavernous and the intercavernous sinuses and the left ophthalmic vein. A large amount of pus was present in the left orbit. Smears showed streptococci.

S. T. 72. A 15-year-old boy had squeezed a comedo at the right angle of the mouth six days before entering the hospital. The next day swelling was present in the upper lip, progressively increasing until the entire upper lip and right face, including the eyelids, were much swollen and the lip red and tender. When admitted his temperature was 101.8 degrees and pulse 112. The white blood count showed 19,600 cells, 89 per cent polymorphonuclear leucocytes. The next day there was marked right exophthalmus and the cellulitis was worse. Slight neck rigidity was present and the spinal fluid examination disclosed 4,160 cells, 90 per cent polymorphonuclear leucocytes present. No micro-organisms were found in the smears. The right angular vein was ligated. Bilateral exophthalmus developed and bilateral Kernig and Brudzinski reactions were elicited. The pulse rate increased to 172, the temperature to 107 degrees, terminally, the next day. At necropsy septic thrombi were found in each cavernous sinus. The other sinuses were clean. There was a small amount of purulent exudate at the base in the pituitary fossa, along the sylvian fissures and in and around the cavernous sinuses.

S. T. 73. A male aged 42 noted a small infected area on his nose five days before he was brought to the hospital. The day after the appearance of this small superficial infection the upper lip was swollen, soon followed by swelling about his left eye. Two days later there was similar swelling about the right eye. Chills and fever now appeared and the day before he was admitted to the hospital in extremis he was delirious. At cisternal puncture the pressure was 200 mm. of water and turbid fluid containing 950 cells per ccm., chiefly lymphocytes, was withdrawn. Globulin was increased to 1-plus and a smear made showed gram positive rods and cocci. He lived but one hour after admission to the hospital. Both cavernous sinuses contained suppurating thrombi and a small amount of purulent exudate was present on the inferior surface of the left temporal lobe extending along its sulci. Coronal sections disclosed no further pathology. Smears from the nasal ulcer, the sinuses and the pleura showed staphylococci.

S. T. 74. A white male of 42 noted a sore on his nose five days before he was admitted to the hospital. The next day the upper lip was swollen and later the left eyelids. Two days later the right eye area was swollen and chills and fever appeared. The day before admission he was delirious and when admitted in coma he was in extremis. The spinal fluid pressure at cisternal puncture was 200 mm. of water and contained 950 cells, chiefly lymphocytes. In smears made from the spinal fluid, gram positive rods and cocci, staphylococcic in form were found. He died within an hour of his entrance to the hospital.

Both cavernous sinuses were thrombosed. A small amount of purulent exudate was present on the inferior surface of the left temporal lobe. A nasal ulcer and bilateral empyema and bronchopneumonia were present.

S. T. 75. A white male aged 48 was admitted to the hospital in coma with the history of the appearance of a furuncle at the right anterior nares six days previously. Poultices had been applied for two days with its rupture resulting. The second day before his admission the right orbital region began to swell and became reddened and very painful, and severe head pain appeared in the right frontal region. The day of his admission he became irrational but had no convulsions. At admission his temperature registered 106.6 degrees, the pulse 140, respirations 32 and blood pressure 110 60. There was right conjugate deviation of the eyes with ecchymosis and ptosis of the right eyelid. The right side of the face was swollen and indurated. The lips and right side of the neck to the subauricular region were swollen. Positive Kernig reactions bilaterally and a left Brudzinski were elicited. Death occurred seventeen hours after entrance to the hospital.

Both cavernous sinuses contained soft sanguino-purulent material. The lateral basal sinuses and middle ears were normal. The longitudinal sinus contained dark blood. At the tip of each temporal lobe was a 2 x 1.5 cm. area of thick, dry, purulent material extending along the sylvian fissures.

The following brief histories are examples of twelve instances in which miscellaneous and multiple sinus thrombosis were presented.

S. T. 76. Both cavernous and each lateral sinuses were found thrombosed in the brain of a negro boy of eleven. Practically the entire surface of the brain was covered with thick, greenish, purulent exudate, especially localized in the longitudinal fissure and over the lateral and inferior surfaces of the right temporal lobe. The left middle ear and mastoid were negative.

The history was given on his admission to the hospital of a painful discharging right ear for one and one-half years. For two weeks there had been more ear pain than usual and chills and fever had been present. The day he was brought to the hospital he had fallen on account of dizziness and a profuse purulent discharge from the right ear was present. His blood pressure was 98 52. Neurological examination disclosed slight elevation of the nasal side of the right optic disc, vertical nystagmus and a peripheral right facial paresis. Movements on the right side were ataxic; all deep reflexes were decreased and bilaterally positive Kernig and Brudzinski reactions were elicited. A smear from the ear discharge showed streptococci and culture growth diphtheriod forms. The spinal fluid pressure was over 350 mm. of water and contained 52 cells, 11 per cent polymorphonuclear leucocytes per ccm. A mastoidectomy was done and, the right lateral sinus being found thrombosed, the right internal jugular vein was ligated. Death occurred the fourth postoperative day.

S. T. 77. Bilateral cavernous and both superior petrosal sinuses were found to contain suppurative thrombi in a white female of 22, who gave a history of pain in the left side of her head for ten days, which had become more severe and accompanied by vertigo and nausea the day she came to the hospital. A left otorrhea had been present as long as she could remember, with increasing deafness in that ear the past few years. A mastoidectomy at the age of 11 had given no relief. The second night in the hospital she suffered excruciating pain all night and a radical left mastoidectomy was done. Two days later the white blood cell count was 12,800 with 78 per cent polymorphonuclear leucocytes and albumen and acetone (2-plus) were found at urinalysis. The blood pressure was 112 75. Thirteen days after the mastoidectomy complaints were offered of blurring of vision of the left eye and neck pain. The median margins of the disks were indistinct and ataxia and dysmetria were present in the left upper extremity. The white blood count at this time was 19,000 with 68 per cent polymorphonuclear leucocytes. No cells were seen at the spinal fluid examination and sugar reduction was shown with four drops of fluid. The mastoid area was re-examined but neck stiffness increased and ten days later increasing coma appeared. Both eyes were swollen and red and the right eyeball diverged. The third day after this, left hemiparesis and bilateral ptosis developed. There was bleeding from the left ear and nostril. The white blood count was 21,000, 90 per cent polymorphonuclears. The Queckenstadt sign was normal with pressure on the right jugular vein, but no response resulted with pressure on the left side. The spinal fluid

colloidal benzoin curve reported was 110,003,333,332,200. The blood sugar content was 125 and nonprotein nitrogen 27 mgm. per 100 cc. of blood. The Wassermann reaction was negative. Death occurred the 35th day of her hospital residence and 32 days postoperative.

Brain examination showed thrombus formation in the veins about the base and purulent exudate in the cisternal space extended into the sylvian and rolandic fissures, more on the right. There were no thrombi in the lateral or sigmoid sinuses. Suppurative thrombi were present in both cavernous sinuses extending into the superior petrosal sinus on the right side. There was also thrombosis of the left superior petrosal sinus. The chamber of the left sphenoid sinus contained mucopurulent exudate, but there was no direct connection with the cranial cavity. The left tympanic cavity was filled with mucopurulent exudate and its wall was eroded into the eustachian tube. A sanguino-purulent area with abscess formation was present in the intratemporal region. The ethmoid sinuses were negative. Several septic pulmonary infarctions were found. The smears from the cavernous sinus thrombi showed staphylococci.

The right cavernous and the right petrosal sinus were found thrombosed in three instances:

S. T. 78. A white male of 48 died seventeen hours after his admission to the hospital because of pain and swelling of the left eyelids, pain in the neck, and inability to sleep, all of which had been present four days. Two days prior to the onset of these symptoms he had expressed some comedones on his nose. At examination the temperature was 98.8 degrees, pulse 160, respirations 28 and blood pressure 140 90. There was marked edema of the left eyelid, injection of the sclera and left exophthalmus. There was some lid discoloration and beginning edema of the right eyelid. The blood examination disclosed a hemoglobin of 100 per cent, 5,200,000 red blood cells and 7,640 leucocytes, 88 per cent polymorphonuclears. Examination of the brain showed thrombosis of both cavernous sinuses with extension into the ophthalmic veins, mostly in the left.

S. T. 79. Four days after squeezing a small furuncle under the left nostril, a 40-year-old white male was admitted to the hospital mentally clouded, with swelling and ecchymosis about the left eye and moderate edema about the right eye. The pulse rate was 96 and temperature 100.4 degrees, increasing to 108 degrees terminally two days later. Brain examination disclosed complete purulent thromboses of both cavernous sinuses and each ophthalmic vein. Hemor-

rhagic, purulent, basilar leptomeningitis was present and an extradural abscess with yellow purulent exudate in the midfossa and about the cavernous sinuses extended into the left orbit.

S. T. 80. A two-months-old male baby was admitted to the hospital semi-moribund with swelling about the eyes, more marked on the right side, and died 19 hours later. The extremities were slightly spastic. The spinal fluid was negative. The history was given of swelling of the right side of the face three days previously and swelling about the right eye and then the left and stiffness of the neck the day before his hospital admission. Left myringotomy resulted in no discharge. Examination of the brain disclosed the presence of a diffuse subdural blood clot over the right cerebral hemisphere with a hemorrhagic meningitis underlying. The right cavernous and right superior petrosal sinuses contained a recent purulent thrombus. Both middle ears were suppurative, more on the left.

S. T. 81. A boy of 12 was admitted to the hospital because of pain behind the right ear for the previous 24 hours. Both ears had been discharging profusely for two months and middle ear disease had been present for five years. Marked neck rigidity was present when he was examined, the reflexes were equal and normal. The white blood cell count was 45,200 with 84 per cent polymorphonuclear leucocytes and the hemoglobin 80 per cent. Filament forms were 38 per cent and nonfilament forms 62 per cent. The next day the white cell count was 27,550 with 92 per cent polymorphonuclears. Filament forms were 36 per cent and nonfilament forms 64 per cent. The spinal fluid was under much increased pressure and contained 8,790 cells, mostly polymorphonuclears. Globulin was 3-plus and no sugar reduction occurred with 30 drops of fluid. Gram stain of the fluid smear showed diplococci. The colloidal benzoin curve was 123,333,000,313,333. Death occurred the third day of hospital residence.

Thick green exudate at the base of the brain extended from the optic chiasma to the spinal nerves, but no localized meningitis or abscess formation was present. Purulent thromboses were found in the right cavernous and right inferior petrosal sinuses. The petrous portion of the temporal bone was necrotic and softened.

S. T. 82. An 8-year-old white boy was brought to the hospital with a temperature of 105 degrees and pulse of 132, following nine days of fever and cough and measles of six days' duration with a right otorrhea for three days. The right eye protruded and the right ear was filled with pus. The white blood cells numbered 16,000

with 70 per cent polymorphonculear leucocytes. The urine was negative. The spinal fluid was clear and contained 36 cells per ccm., 50 per cent polymorphonuclears and 50 per cent leucocytes. A right mastoidectomy was done the day after he was admitted to the hospital and he died two days later.

In the right cavernous sinus a fibrinous purulent greenish-yellow thrombus, thick pus in its lower and posterior portion, extended for 4 cm. into the superior petrosal sinus. The superior cerebellar veins were engorged. Bilateral otitis media, more extensive on the left side, was present.

S. T. 83. Early exitus after development of cerebral symptoms also occurred in a male 27 years of age who was admitted to the hospital moribund, dying in four and one-half hours. The temperature was 105 degrees, pulse 104 and respirations 42. There was swelling of the left side of the nose and the eyes were swollen nearly shut. A week before a furuncle had appeared in the left nares. No trauma to the lesion had occurred, but the morning of his hospital admission he became suddenly drowsy and quickly lapsed into coma.

Brain examination disclosed free pus in the right cavernous sinus and bloody serous exudate in both cavernous sinuses. Some thin pus was present at the base and a subdural hemorrhage had extruded over the left cerebral hemisphere. The left middle cerebral vein was distended with dark semifluid blood. The left eye tissues were hemorrhagic and the ethmoid and sphenoid sinuses contained clear serous exudate.

S. T. 85. Following removal of a tooth two weeks before admission to the hospital, neck swelling had occurred in a white female of 23. She had been delivered normally two weeks before the dental extraction. When admitted to the hospital she was semicomatose and septic. A marked cellulitis of the neck contained drains on either side. Marked bilateral exophthalmus was present, the fundal veins were dilated and the discs slightly choked. The conjunctivae were reddened and edematous. The spinal fluid was cloudy and contained 2,400 cells, 50 per cent leucocytes per ccm. Death occurred the second day after admission to the hospital.

At necropsy the socket of the extracted first right lower molar was found to be negative. There was extensive cellulitis of the left side of the neck and the parotid gland was saturated with pus. There were two draining incisions in the left side of the neck and one below the right mastoid. To the left of the falx cerebri the brain was covered with 150 cc. of pale, greenish, thick pus. The ventricles

contained turbid fluid. The left petrosal and cavernous sinuses were filled with thick green pus, which exuded along the left optic nerve into the retrobulbar region, where it was quite extensive, 8 cc. being contained in the left orbit. The right petrosal sinus contained no pus, but the right cavernous sinus was filled with the same thick green pus. There was none in the right retrobulbar region. The sphenoid sinus was filled but the remaining sinuses, the middle ears and mastoid cells were free. Smears showed bacillus pyocyaneus present.

S. T. 86. Each inferior petrosal sinus was found thrombosed from its origin at the cavernous sinus to the jugular bulb in the brain of a white female of 37, who was in the hospital 16 hours. Headache, fever and malaise had been present one week and she had been semicomatose for a day when she was brought to the hospital. The right pupil was irregular and reacted poorly. The left pupil was fixed. Intermittent right-arm spasticity was present. The reflexes were equal and none pathological. The spinal fluid was cloudy and contained 2,000 cells per ccm. Globulin content was 1-plus and 17 drops of fluid failed to reduce Fehling's solution. Smears from the fluid showed pneumococci. The cerebral vessels were found extremely congested and dilated. A large amount of green yellow pus, mostly basal, lay subarachnoid along the cerebral vessels, coming through the Sylvian fissure. Both inferior petrosal sinuses were thrombosed. The wall of the sphenoid sinus was eroded. There was no intracerebral pathology. A pulmonary embolus was present in the left lower lobe.

S. T. 87. An 18-months-old female baby had suffered a "cold" with chills and fever and continued cough a month before admission to the hospital. Improvement occurred; then after two weeks she became worse. When admitted the temperature was 106 degrees R, pulse 170, respirations 32. Both ear drums were bulging. The leucocyte count was 24,000 with 90 per cent polymorphonuclears. Following bilateral myringotomy, improvement occurred for a few days, but death resulted fifteen days after admission.

Diffuse purulent meningitis was present, especially about the basal vessels. The middle ears contained pus, which extended into the mastoid cells, producing bone necrosis. The left sigmoid sinus was thrombosed. The left upper pulmonary lobe was involved by lobar pneumonia with left empyema adjoining. Smears from the meninges showed the presence of encapsulated pneumococci.

S. T. 88. A white male, aged 34, reported a chronic discharging ear for many years when he was admitted to the hospital. Head-

ache had been severe for ten days and fever noted for five days, with the occasional appearance of diplopia. When examined on admission the temperature was 100.2 degrees, the pulse rate 84 and respirations 22 per minute. The right pupil was larger than the left and the right external rectus was weak. The fundal vessels were engorged and tortuous and the right disc presented two diopters of swelling. There was no mastoid tenderness. The roentgenological examination of the right mastoid showed increased density throughout with bone absorption in the region of the lateral sinus and the body of the mastoid with extension toward its petrous portion. The spinal fluid pressure was 380 mm. of water with negative Queckenstedt responses and 30 lymphocytes per ccm. present. The Wassermann reaction on the fluid was 4-plus and Kahn 1-plus. A right mastoidectomy was done 24 hours after admission, at which time mastoid sclerosis and necrosis of the wall of the lateral sinus and of the exposed dura of the temporal lobe were found and cholesteatoma were cleaned out. Evidences of meningeal irritation appeared the next day and jaundice the day following. A blood transfusion was given the day before his death, which occurred the fifth day of his hospital residence with a premortem temperature of 107 degrees.

The entire right transverse sinus contained an antemortem thrombus which was dark red between its knee and the torcular. In the sigmoid sinus the thrombus was more firm and had a purulent tinge. The dura in this region showed an early necrotic and purulent change. The greater portion of the right mastoid and part of the tympanic portion had been removed, exposing the middle ear. In the tympanic cavity, attached to its walls and to its ossicles was a 5 mm. mass of yellow tissue of the cholesteatoma. The interior of the petrous tip showed a slightly hemorrhagic serous exudate. An acute splenic tumor and a gangrenous lung abscess in the right lower lobe at the site of a septic infarct with right-sided empyema were present.

S. T. 84. Erysipelas, involving the left side of the face had been present for four days in a female of 58, otherwise healthy, when she entered the hospital. The left eye was swollen shut. The temperature varied between 103 and 105 degrees the 48 hours she was in the hospital, evidencing increasing septicemia before her death.

Moderate superficial hyperemia of the brain was shown. Each cavernous sinus and the veins of the basilar plexus contained a mixture of blood and pus. The lungs contained multiple septic infarcts. An acute splenic tumor was present and a large hemorrhage in the left abdominal rectus muscle.

CAVERNOUS, PETROSAL AND SIGMOID SINUS THROMBOSIS (34)

Serial No.	Autopsy No.	P. F. No.	Initials	Color	Sex	Age	Distribution
55	5382	6-038	Z. F.	White	F.	40	Lt. cavernous
56	5989	76-614	R. B.	White	F.	22	Lt. cavernous
57	10020	277-747	J. R.	White	F.	18	Lt. cavernous
58	11474	352-807	J. C.	White	M.	26	Lt. cavernous
59	11628	354-604	A. F.	White	M.	5	Lt. cavernous
60	5364	39-876	I. G.	White	M.	33	Rt. cavernous
61	8662	213-755	C. D.	White	F.	14	Rt. cavernous
62	8977	232-559	M. A.	White	M.	14	Rt. cavernous
63	9193	244-593	C. P.	White	M.	51	Rt. cavernous
64	12192	383-501	W. W.	White	M.	22	Rt. cavernous
65	12402	392-444	O. B.	White	M.	42	Rt. cavernous
66	7875	173-392	W. U.	White	M.	11	Bilat. cavernous
67	3352	250-808	E. H.	White	F.	52	Bilat. cavernous
68	3649	258-244	A. R.	White	M.	40	Bilat. cavernous
69	3995	264-896	T. McD.	Black	M.	27	Bilat. cavernous
70	5160	28-009	H. S.	White	M.	19	Bilat. cavernous
71	6359	96-989	P. R.	White	F.	62	Bilat. cavernous
72	7921	177-878	I. M.	White	M.	16	Bilat. cavernous
73	8375	199-054	A. M.	White	M.	42	Bilat. cavernous
74	8675	214-964	J. R.	White	M.	72	Bilat. cavernous
75	12122	380-772	F. M.	White	M.	48	Bilat. cavernous
76	7433	151-174	G. P.	Black	M.	11	Bilat. cavernous Bilat. lateral
77	7998	174-614	E. D.	White	F.	22	Bilat. cavernous Bilat. sup. petrosal
78	8370	199-069	F. S.	White	M.	48	Bilat. cavernous Bilat. ophthalmic veins
79	8835	222-413	B. S.	White	M.	40	Bilat. cavernous Bilat. ophthalmic veins
80	3880	262-136	D. S.	White	M.	2 mo.	Right cavernous Rt. sup. petrosal
81	8381	199-214	J. M.	White	M.	12	Right cavernous Rt. inf. petrosal
82	9881	206-357	G. P.	White	M.	8	Right cavernous Rt. sup. petrosal
83	10057	283-113	F. R.	White	M.	27	Bilat. cavernous Lt. mid. cereb. vein
84	12281	387-598	O. P.	White	F.	58	Bilat. cavernous Basal veins
85	4394	275-302	J. E.	White	F.	23	Lt. sup. petrosal
86	8276	171-155	P. V.	White	F.	37	Bilat. inf. petrosal
87	10320	196-367	M. T.	White	F.	1½	Left sigmoid
88	11706	361-639	J. W.	White	M.	34	Right transverse and sigmoid

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XXXVI

RECONSTRUCTION SURGERY OF THE NOSE*

GEORGE WARREN PIERCE, M.D.

AND

GERALD BROWN O'CONNOR, M.D.

SAN FRANCISCO

Reconstruction surgery of the nose presents a great variety of problems. The manner of dealing with these problems is open to discussion, as no set methods can be put forward for the variable factors involved. Each case requires careful study and analysis, and it is not unusual for the surgeon to change his plan of procedure in the midst of the operation because of unsuspected variations.

No attempt has been made to cover the rich field of historical data on this subject, as the field is too voluminous, but the authors have given their own experience in dealing with each problem. Each case is presented with nonessential history omitted, but with particular attention to plan and management.

The authors wish to stress especially the use of preserved cartilage isografts as described in case 12. They feel that this contribution will remove the danger associated with operations for obtaining rib cartilage, and will simplify the operation for the patient, both as to hospitalization period and discomfort.

Reconstruction of the tip of the nose is seldom a simple problem, as it is usually complicated by partial loss of one or both alæ as well as of the columella. As restoration of the alæ requires provision of epithelial lining as well as covering, the difficulties of the procedure are increased.

Case 1, Fig. 1, illustrates loss of a portion of the nasal tip, left ala and all of the epithelial covering of the columella from a glass cut. The resulting scar was more disfiguring than appears in the photograph.

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*Read before the Western Section Meeting of the American Laryngological, Rhinological and Otological Society, at Santa Barbara, California, January 29, 1938.

Fig. 1 Case 1

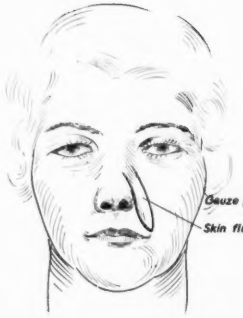

Fig. 2 Case 1

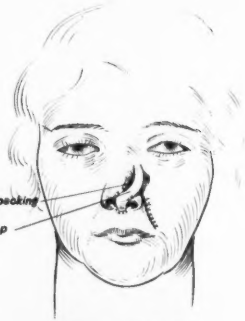

Fig. 3 Case 1

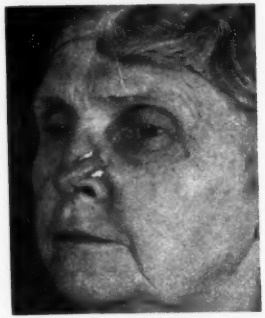

Fig. 4 Case 1

Fig. 5 Case 1

Fig. 6 Case 1

To repair this tissue loss a descending flap was planned in the naso-labial fold and this was extended almost to the left corner of the mouth. The flap lent itself nicely to the repair, as it replaced the tissue loss of the tip and was also carried on down the length of the columella. The resulting scar on the face is negligible, as can be seen in the subsequent photographs, and the advantage of using the facial skin for similar coloring and texture must not be overlooked. The line drawings of Figs. 2 and 3 illustrate the site of the flap as well as the transfer after scar tissue had been excised from the tip of the nose and columella. Fig. 4 shows the flap healed. The pedicle of the flap was cut free after three weeks and the stump returned. Figs. 5 and 6 show the final result.

Case 2, Fig. 7, illustrates a problem similar to that of case 1, but the destruction of tissue as the result of an explosion was more extensive. The alæ were largely destroyed and the stumps and tip were covered with a thin shiny red scar. At the time this photograph was taken the scar of the entire bridge of the nose had been replaced by a full thickness skin graft. For the restoration of the tip and alæ no face skin was available, as that entire area as well as the forehead was badly scarred. A tubed pedicle of small diameter was made parallel to and just above the left clavicle. After healing of the suture lines this pedicle was stepped up at intervals of three weeks and the progress is shown in Figs. 8, 9 and 10. Fig. 8 shows the second step up, one end of the pedicle having been previously implanted under the chin when the other was at the base of the neck. Fig. 11 illustrates the final result and shows how nicely the small tubed pedicle lends itself to the solution of this rather exacting problem.

Case 3. In an explosion a sharp fragment of steel cut off half this patient's nose (Fig. 12). By undermining the skin of the cheek and swinging a flap the upper half of the defect was closed. After healing of the flap was complete, reconstruction was started by burying a strip of costal cartilage 3 cm. long by 5 mm. wide and 3 mm. thick beneath the skin of the cheek. One end of the cartilage rested in the stump of the ala. After four weeks a flap from the cheek was turned over to form the lining of the ala. On its raw surface it bore the implanted cartilage which had become surrounded and fixed by a fibrous envelope. Thus was provided an epithelial lining and a cartilaginous support. The epithelial covering was supplied from a pedicled flap from the forehead, after the Indian method. Fig. 13 illustrates the finished result. The cartilaginous support has main-

Fig. 7 Case 2

Fig. 8 Case 2

Fig. 9 Case 2

Fig. 10 Case 2

Fig. 11 Case 2

Fig. 12 Case 3

Fig. 13 Case 3

tained a well opened airway and there has been no collapse over a period of years.

Case 4. As the result of an automobile accident, this patient sustained the loss of half the right ala (Fig. 14). When the wound healed the airway on that side was almost completely obliterated and the remaining opening just admitted an ordinary pin. For the reconstruction of this ala a descending flap in the naso-labial groove was selected, a flap of the type used in case 1.

At operation all scar tissue was dissected out and the defect was reconstituted. By using a pattern of sheet lead a proper flap was raised and so folded at the tip that the covering, lining and border of the ala were provided. Figs. 15 and 16 illustrate the procedure, while Fig. 17 shows the reconstructed ala with airway three months after operation.

Case 5. This patient's case was of unusual interest, as it presented a complicating clinical problem. She had suffered for twenty-five years from lupus vulgaris involving the skin of the nose. The characteristic appearance of lupus vulgaris can be noted in Fig. 18. The nose had shrunken to a mere nubbin on the face. Cure of the lupus vulgaris was the prime necessity before reconstruction. This was accomplished by carefully dissecting off all the affected skin and then covering the raw surface with a split skin graft by the epithelial outlay method. Ten days later the splinting wax was removed and it was evident that a complete take had been obtained, as is to be expected in this type of skin graft. It is interesting to note here that there has been no recurrence of the lupus vulgaris after eight years. In other words, lupus of twenty-five years standing was cured in ten days. Fig. 19 shows the healed skin graft. Three months later reconstruction was started. Flaps were turned in from the cheeks to line the alæ, the skin graft was dissected off and covering was provided by a pedicled flap from the forehead. The forehead defect was filled with a full thickness skin graft (see Figs. 20 and 21). The profile as shown in Fig. 21 could be improved by implantation of costal cartilage in the bridge and with a columellar post of the same material.

Case 6, Fig. 22, shows a destruction of almost all of the skin and subcutaneous tissue of the nose, due to implantation of foreign material. The mucosa of the left airway showed a perforation at the lower border of the nasal bones. At the time of operation the edges of the opening through the mucosa were trimmed and inverted with a purse-string suture of plain 000 catgut. The thin epithelial cov-

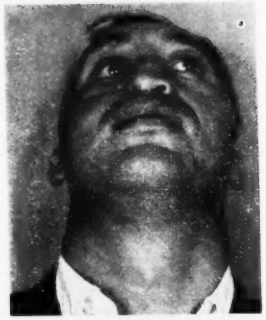

Fig. 14 Case 4

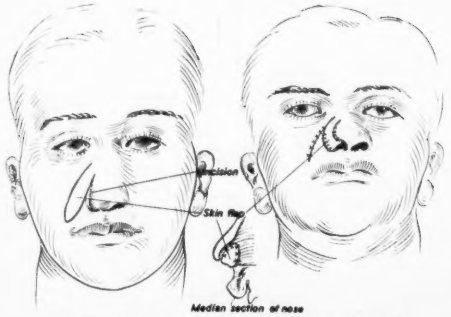

Fig. 15 Case 4

Fig. 16 Case 4

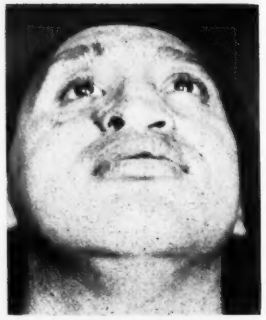

Fig. 17 Case 4

Fig. 18 Case 5

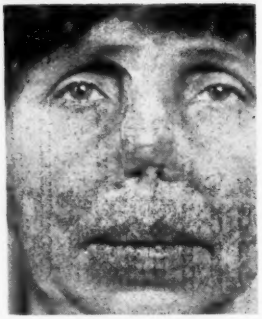

Fig. 19 Case 5

Fig. 20 Case 5

Fig. 21 Case 5

ering of the scar was dissected off and a flap from the forehead of the Indian type was brought down to cover the area. Final result is shown in Fig. 23.

Case 7, Figs. 24 and 25, show the loss of the nasal tip and portions of both alæ from a cut by flying glass. The resulting scar constituted a marked cosmetic deformity and greatly reduced the size of the nostrils with consequent diminution of aeration. The problem here is magnified by the difficulty of reproduction of the size and shape of the nostrils, together with provision of proper epithelial lining. This lining was obtained at operation by swinging flaps of skin from the stumps of the alæ, after the original defect had been reconstituted. Covering was provided by a forehead flap of the Indian type pedicled on the angular artery. The remaining raw area of the forehead was covered with a full thickness skin graft.

Figs. 26 and 27 show the result shortly after the return of the pedicle. The visible scars have since faded, leaving little evidence of the injury.

Case 8, Fig. 28, illustrates the condition of a patient who came eight months after he had been kicked in the face by a horse. As will be seen, the nasal cavity is open, with destruction of the nasal bones and partial destruction of the vomer and quadrilateral cartilage. For the repair of this defect lining, covering and support were needed. At the first operation flaps of skin were turned in from the sides of the defect. A pedicled flap from the forehead provided the covering. Three weeks later the pedicle was cut and returned to the forehead. Ten weeks later a support of costal cartilage was inserted through the scar in the glabellar region. Fig. 29 shows the result.

Case 9 shows a patient who had lost one entire side wall of the nose in a fall from a scaffold (Fig. 30). We have again the problem of lining and covering. In this case a somewhat different solution was arrived at. A flap was planned on the high forehead, to be pedicled on the temporal artery. Beneath the extremity of this planned flap a skin graft was done by the epithelial inlay method through a single two-inch incision. Ten days later the wax was removed, revealing a complete take of the skin graft, and thus an epithelial lining was provided for the flap. Three days after the removal of the wax, the projected incisions were made and the pedicled flap was brought down and sutured to the nose (see Fig. 31). The flap remained for three weeks and was then cut at the appropriate place,

Fig. 22 Case 6

Fig. 23 Case 6

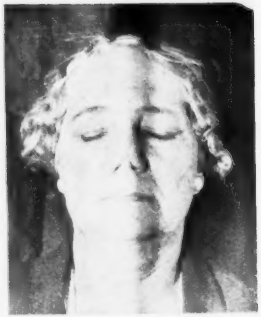

Fig. 24 Case 7

Fig. 25 Case 7

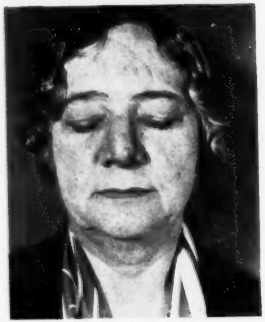

Fig. 26 Case 7

Fig. 27 Case 7

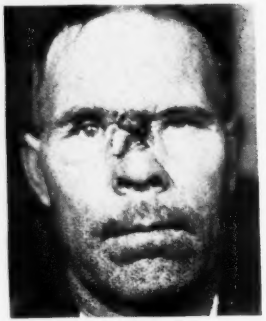

Fig. 28 Case 8

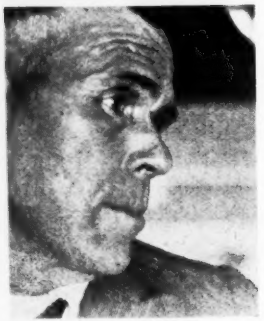

Fig. 29 Case 8

trimmed and sutured into the cheek. The remainder of the pedicle was returned to the forehead, the defect there remaining having been previously covered with the epithelial inlay, as noted above. The ultimate result is shown in Fig. 32. The flap from the scalp is of sufficient thickness that no cartilaginous support was needed.

Case 10 is a loss of the entire columella and lower end of the septum. Here again was used the small tubed pedicle, made parallel to and just above the clavicle (Fig. 33). This was stepped up to the chin, upper lip and finally to the nose at intervals of three or four weeks. Figs. 34 and 35 show the final stage.

Case 11, Fig. 36, illustrates the method of treatment employed in a case of complete atresia of the right vestibule. This man fell on a sharp pointed steel rod and, when healing had occurred, the contracting scar had closed the airway, with only a minute opening remaining. The author has seen efforts made to correct such a condition by cutting through the band of scar and then packing with rubber dam or insertion of a rubber tube, the operator hoping that epithelialization of the raw surface would occur. Some epithelialization does take place, but the procedure is futile, as annular scars contract tremendously and the deformity recurs.

In the case illustrated above, all of the scar tissue was dissected out carefully. A previously prepared cap splint for the upper teeth with a projecting german silver wire ending in a narrow perforated plate was cemented onto the upper teeth. At its center the wire carried a binding post to admit of adjustment of the plate. Dental modeling wax was accurately molded to the defect and included the perforated plate at the end of the wire. A split skin graft was taken from the arm and wrapped around the wax, and this was then placed in firm apposition with the entire raw surface and the binding post screw was set. The patient wore the apparatus for eighteen days. It was removed and the entire raw area was found to be epithelialized. As the scar tissue was minimum, very little contracture took place, and the patient was provided with an ample airway.

Case 12 is a patient who came to us eight months after a submucous resection of the septal cartilage. There had been a gradual sinking in of the nasal bridge and the tip lacked proper columella support (Figs. 37 and 38). The author has tried all the numerous recommended foreign bodies for implantation and has come to the conclusion that none of these compare with costal cartilage. For the past five years we have used isografts of costal cartilage which have been preserved. Most bodily tissues when transplanted must

Fig. 30 Case 9

Fig. 31 Case 9

Fig. 32 Case 9

Fig. 33 Case 10

Fig. 34 Case 10

Fig. 35 Case 10

Fig. 36 Case 11

be autografts and this even applies to skin. However, costal cartilage differs in cellular structure and reactions and has been found adaptable for use as isografts. The isografts are prepared by cleaning off all soft tissue, including all of the perichondrium, and they are then placed in a solution consisting of one part of aqueous merthiolate and four parts of normal saline solution and kept in an ice box. They are transferred to a fresh solution after three days and again after a week. They are then cultured for sterility. We have found that they show less tendency to bend or curl, that they heal more firmly to the surrounding tissue and that they resist infection better than autografts of the same material. The advantages of isografts are enormous, in that the painful and moderately dangerous operation is obviated and hospital stay is reduced from at least a week to one day. We have kept the material in solutions as long as one year before using. We have employed this material in one hundred and eighty-two cases, with no untoward results. This method of preserving and using isografts of costal cartilage was first reported by the authors on January 3, 1935, at a regular St. Mary's Hospital Staff Clinical Meeting at San Francisco, California.

We are carrying on research work at present to determine the viability of these cells, their biochemistry and their ultimate fate.

In the case shown above, a mid-columella incision was made, the skin tunneled over the bridge of the nose, one piece of refrigerated rib cartilage shaped for the bridge and another for the columella post, and these were then inserted and the wound closed. Figs. 39 and 40 show the result.

Case 13 is one of a saddle nose, the result of an old fracture. Cartilage was used in the same manner as in case 12. The result is shown in Fig. 42.

Case 14 is another patient who developed a deformity similar to the one shown in case 12; that is, following a submucous resection of the septal cartilage. A study of the contours of this patient's face (Fig. 43) decided us to advise her to have a resection of the bridge of the nose rather than to build up with cartilage. The patient disliked the rather large nose she had before the submucous resection. At operation incisions were made in each vestibule and the hump was sawed off. The nasal processes of the superior maxillæ were sawed through at their bases and fractured inward. This narrowed the nose and with the lowered bridge gave a pleasing contour. (Fig. 44.)

Fig. 37 Case 12

Fig. 38 Case 12

Fig. 39 Case 12

Fig. 40 Case 12

Fig. 41 Case 13

Fig. 42 Case 13

Fig. 43 Case 14

Fig. 44 Case 14

Cases 15, 16, 17 and 18, illustrate a common problem of the reconstruction surgeon. Many people come who have so worried over the shape of their noses and who have thought so constantly about that appendage that the matter has become an obsession. A considerable number actually develop an inferiority complex which colors their whole lives. They all will admit readily that reason tells them that they are attaching an importance to the nose out of all proportion. Some with slight irregularities of contour have, in an escape from entirely unrelated problems in their lives which they will not face, fixed on their noses as the source of all their misfortunes, and thoughts of that appendage fill their every waking moment. Such patients should not be encouraged in their desire for operation; they should be avoided, and should be referred, if possible, to a psychiatrist until they obtain a proper mental proportion. Operations on these invite trouble. However, the great majority of those who come with definite and very noticeable variations from the ideal type of nose should be operated upon if they are advised that the procedure is a real operation and if too much is not promised. These patients tend to be hypercritical of small variations in contour after the operations and should be discouraged of too close and constant examination of their faces before the mirror. The authors find that a good cure for this tendency is to call their attention to the very obvious differences in the opposite sides of their faces. The patients generally admit with surprise that they had never noticed these differences before.

These operations, if properly done, do markedly improve the appearance of many people, and we constantly see an increased confidence and enjoyment of life as a result of them.

The work must be carefully and deftly performed, with a minimum of trauma, and a detailed study must be made of every phase of the problem. The old day of merely rasping off or sawing off the hump is gone. The author is convinced of the thoroughness and superiority of the technique of Joseph for this operation and follows in general his plan and principles.

Case 15, Fig. 45, shows a hump of the nasal bridge and also a large tip with a too large columella and consequent unsightly nostrils. Fig. 46 indicates the result of correction.

Case 16, Fig. 47, shows a similar problem of hump and columella. In addition, the upper lip is encroached upon by the columella, making it appear too short. Fig. 48 illustrates the completed correction.

Fig. 45 Case 15

Fig. 46 Case 15

Fig. 47 Case 16

Fig. 48 Case 16

Fig. 49 Case 17

Fig. 50 Case 17

Fig. 51 Case 17

Fig. 52 Case 17

Fig. 53 Case 18

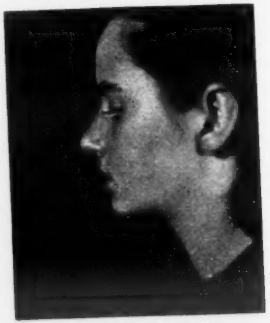

Fig. 54 Case 18

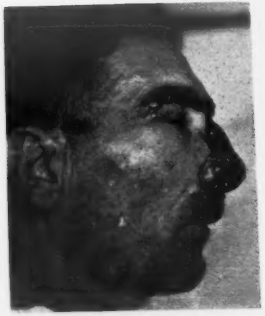

Fig. 55 Case 19

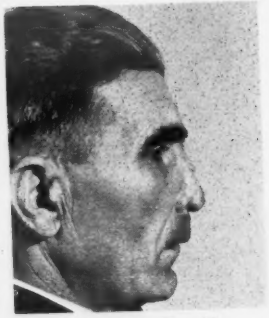

Fig. 56 Case 19

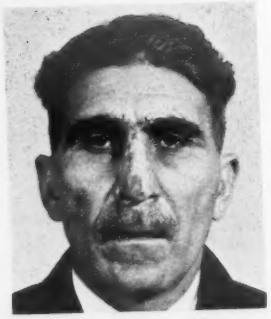

Fig. 57 Case 19

Case 17, Figs. 49 and 50, show again the long nose with hump and also a broad tip. Figs. 51 and 52 show the shortened nose, straight bridge, shortened columella and narrowed tip, all of which are essential for a successful result.

Case 18, Fig. 53, illustrates an extreme type of nasal anomaly. The hump, the heavy drooped tip and grossly exaggerated columella are entirely out of proportion to the rest of the face and make the patient look years older than her actual age of 16 years. Fig. 54, taken after the operation, shows a nose in keeping with the face and fitting to her teens.

Case 19 offers an unusual and interesting problem. This patient was attacked with a butcher knife and his nose cut off. It was actually cut off and fell to the floor, severed from the glabella through bone, cartilage, skin and mucous membrane to the base of the columella. He pursued his assailant but could not capture him. He returned and picked up the nose. The patient was transported several miles to Dr. Edw. C. Faulkner of Rio Vista, California, who sutured the nose to the face one hour after it was severed. Six horsehair sutures were used, one at the glabellar region, one at each side to the cheek, one in each ala, and one at the base of the columella. He was then transported fifty miles, where he came under our care. There was little we could do except to arrange an electric globe near and above his nose so as to maintain a constant and gentle warmth. The patient showed a remarkable stoicism and co-operation, scarcely moving for several days. The nose turned very dark and numerous blebs appeared on the skin surface during the first four or five days. Gradually the nose became pink, first in spots and later throughout. The blebs were removed and all the superficial epithelium exfoliated. There were spots of slight and partial necrosis in some areas of the corium, but the nose remained viable. Figs. 56 and 57 show the condition six weeks after severance of the nose. Since then the appearance has further improved, so that there remains little evidence of the injury.

The foregoing cases present a variety of the problems which the reconstruction surgeon is called upon to solve. The authors present them in the hope that they may stimulate further improvement in planning and technique.

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XXXVII

LARYNGECTOMY—ONE STAGE*

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Early attempts at surgical intervention in the treatment of carcinoma of the larynx produced, as in other fields, an appallingly high mortality rate. As we trace the development in this field of endeavor through the years from the pioneers—Ehrmann (1844), Buch (1855), Cohen (1868), Billroth (1873), Gluck (1881), and many others—to the moderns—Lewis, Jackson, MacKenty, Crile, Woods, New, Orton, Clerf, and a host of others, one fact stands out: Surgery has been proven the treatment of choice in cases of carcinoma of the larynx, provided diagnosis is made in time. As Clerf has pointed out in a recent article, "The patient, the general practitioner and the laryngologist must co-operate if the number of inoperable cases of cancer of the larynx is to be decreased."[1]

With the ideal diagnostic facilities existing today and the medical profession alert to the teachings they must present to the medical students now receiving training under our guidance, we should be increasingly able to detect, correctly diagnose and treat new cases of laryngeal carcinoma. Late diagnosis as the fault of the patient is lamentable but understandable. He dismisses the warning symptoms as unimportant, or attributes them to other causes. Or, fearing the dreaded diagnosis, he postpones seeing his doctor until forced to do so by his condition which has become alarming and can no longer be denied. On the other hand, late diagnosis as the fault of the doctor is no longer to be tolerated. X-ray, radium and diathermy all have their place, but they should not be experimented with in an attempt to clear up a condition until that condition is truly diagnosed and the method of treatment decided on. It should be borne in mind that a positive Wassermann in a case of hoarseness does not rule out cancer. Nor, inversely, does a positive pathological report rule out lues. Likewise it should be stressed again and again that the old idea of a definite "cancer age" (middle age) is an exploded theory.

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*The Waitman F. Zinn Bronchoscopic Clinic, Mercy Hospital, Baltimore, Md.
Presented before the Southern Section of the American Laryngological, Rhinological and Otological Society, Atlanta, Ga., January 24, 1938.

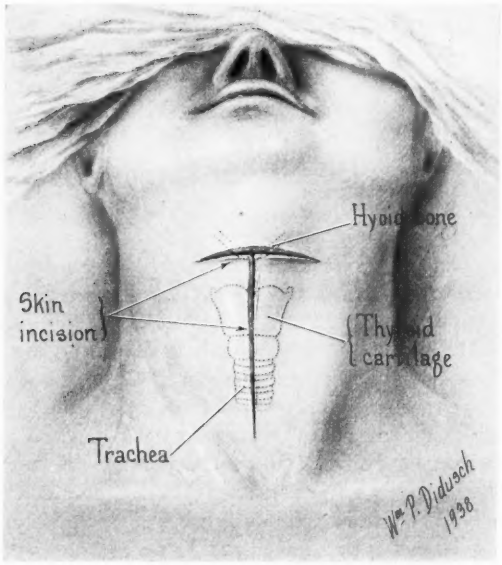

Fig. 1. Skin incision.

The symptoms presenting themselves in carcinoma of the larynx are at times confused by the presence of other complications. Hoarseness is a constant in more than 90% of the cases and is usually first to appear. Cord immobility, always present eventually, is next in the typical picture. The lesion, in the incipient stages, will be single and flat in appearance with no inflammation about it. There will be no pain in the early stages. Syphilis, tuberculosis and cancer may all be present within the same larynx at one time, though this is not common. Locating one disease does not, therefore, rule out either or both of the others.

Granted, then, that the diagnosis of laryngeal cancer has been made. Where is the lesion located? If within the larynx itself, arising from the cords, the ventricles, the ventricular bands, the interarytenoid and subglottic areas, it is classed as intrinsic. If it is found at the epiglottis, the arytenoids, the aryepiglottic folds, the pyriform sinuses or pharyngeal surface of the cricoid cartilage, it is extrinsic in type. The latter are extremely bad operative risks since

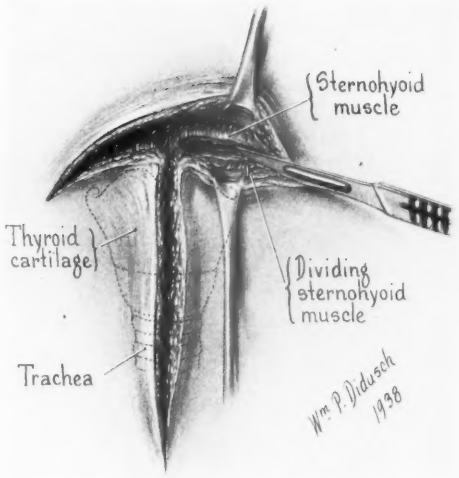

Fig. 2. Dividing the sternohyoid muscles.

there is likely to be a metastasis through lymphatic extension. If not too far advanced, some few of these cases may yield to surgery followed by radium or x-ray. On the other hand, the intrinsic class is a good risk and should have the advantage of early surgery following closely a positive biopsy report. Biopsy should never be omitted as the final step in the routine examination of the patient. The clinical aspect is important and should be considered jointly with the microscopic findings.

Surgical technique varies with the surgeon. Some have obtained excellent results with thyrotomy. I feel, however, that because of the difficulty in judging the extent of the disease in the underlying tissues, the patient should be given the advantage of removal of all cancer tissue by total extirpation. Whether the operation be one-stage or two is again a matter of choice with the operator. That method which produces the best results should be used. Personally, I favor the one-stage laryngectomy.

To the late John E. MacKenty we are indebted for the most comprehensive reports of operative technique in the field of laryngectomy. My debt to him I sincerely and humbly acknowledge.

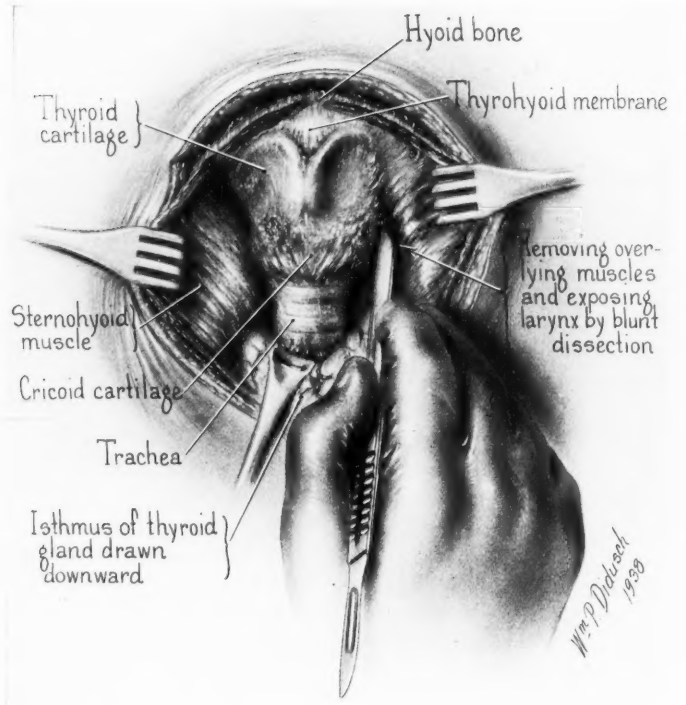

Fig. 3. Removal of overlying muscles and exposure of larynx by blunt dissection.

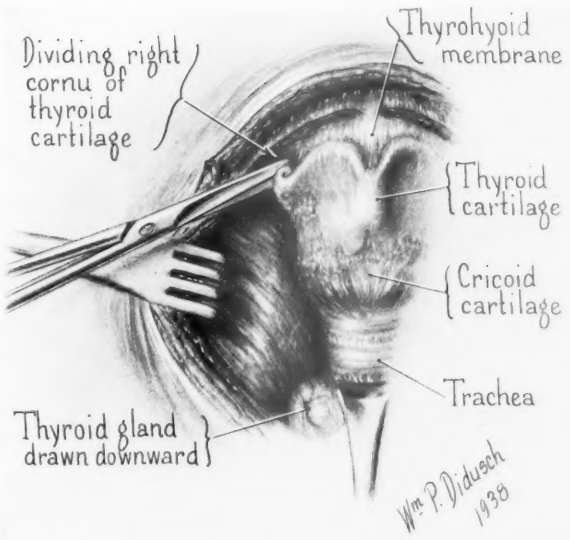

Fig. 4. Division of right cornu of thyroid cartilage.

Preparation of the patient for the operation is important. He should be admitted to the hospital at least four days prior to the operation and should have complete physical examination and laboratory work-up. Special mouth hygiene should be instituted and a sterile diet should be administered the day prior to the operation. The choice of anesthetic and its administration are of utmost importance. My preference is for nitrous oxide and drip ether, following avertin. All colonic irrigations should be stopped 24 hours prior to the operation. The patient is shaved and the avertin administered before he is taken to the operating room.

A T-shaped incision is made as illustrated in Fig. 1. The cross stroke of the T is made slightly above the upper border of the thyroid laminæ, about at the hyoid bone. The downstroke extends well below the second tracheal ring. All bleeding points should be quickly ligated by means of fixed sutures. The process of skeletization of the larynx begins with the division of the sternohyoid muscle. (Fig. 2.) Blunt dissection proceeds from the anterior part of the thyroid cartilage posteriorly and downward to the second tracheal ring. (Fig. 3.)

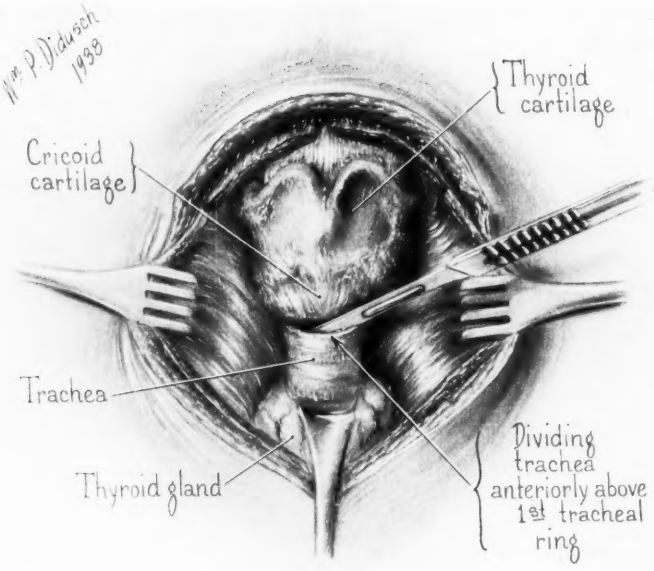

Fig. 5. Division of trachea anteriorly above first tracheal ring.

The right and left cornu of the thyroid cartilage are now divided. (Fig. 4.) The thyroid gland is drawn downward in preparation for the incision of the trachea. It is particularly important that all bleeding points be located and ligated before the next step is taken so that no blood may enter the lungs. Retractors are inserted on each side of the larynx and the trachea is severed anteriorly above the first tracheal ring as shown in Fig. 6. The flap of mucous membrane on the posterior wall of the trachea must be carefully preserved. (Fig. 6.) Suction takes up any oozing that may occur and a tube, such as shown in Fig. 7, is inserted into the trachea, the disc of the tube being over the cut end of the open trachea. This tube, as it is designed, has several advantages worthy of notice. There are two branches, one for the continuance of the general anesthetic and one for the administration of oxygen, if needed. It may be made to fit into any larynx by the use of layers of bismuth gauze around the end. It leaves the operative field clear and assures an absence of seepage from the field into the lungs.

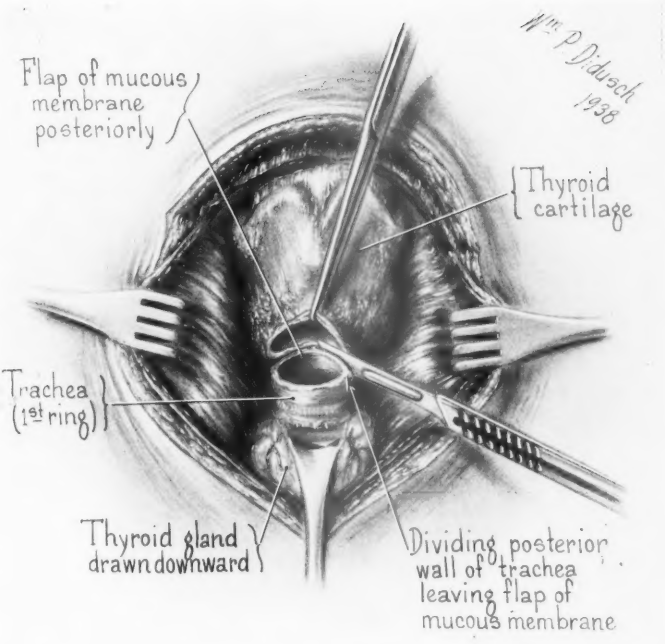

Fig. 6. Division of posterior wall of trachea leaving flap of mucous membrane.

The larynx is now drawn upwards as the posterior is dissected from the esophagus up to the arytenoids. (Fig. 7.) It is then allowed to fall back in place and the thyrohyoid membrane is incised, opening the hypopharynx at a point determined by the extent of the disease. (Fig. 8.) The epiglottis is usually left intact unless the disease is particularly widespread. Dissection of the larynx is now completed and it is removed for macroscopic and microscopic examination. Gauze, saturated with mercurochrome, is packed into the hypopharynx, the pharynx and the mouth, as shown in Fig. 9. The transverse, or upper part of the T, closure of the hypopharynx is effected by suturing through the thyrohyoid membrane. (Fig. 10.) The gauze is removed through the mouth. The naso-esophageal feeding tube is directed into the esophagus before the lower stitches of the longitudinal closure are tied. (Fig. 11.)

The tracheal stump is now securely anchored with three stay sutures supported on three medium sized buttons which prevent tearing the skin. (Fig. 12.) These are removed within 48 hours. Three sutures are used to close the opening above the trachea and ample opportunity for drainage is allowed in that no attempt is made to bring the edges of the incision closely together. The suturing of the tracheal stump is now completed by fastening it smoothly to the skin around the opening. (Fig. 13.) Gutta-percha drains are inserted at each end of the transverse incision and at the base, just below the tracheal opening. Two double tube drains, open at distal and proximal ends to allow free irrigation of wound pockets, are inserted slightly above the tracheal opening. Additional gutta-percha drains are inserted into stab wounds on either side of the trachea. The placing of the drains is very important and aids greatly in controlling any infection which may extend downward along facial planes. Iodoform gauze is packed around the tube drains. (Fig. 13.) A laryngectomy tube wound with gauze and smeared with bismuth paste is inserted into the trachea (Fig. 14) and the dressing applied. A rubber sheet is fitted around the cannula to prevent wetting by tracheal secretions.

The after-care of the patient is most important and the surgeon himself should do all dressing of the wound. Sloughing is a good sign for it prevents hidden infection from getting an uncontrollable start. The drains remain in place for about one week and are first cleaned on the second or third day. Saline solution is forced through the tubes by a suction apparatus as often as is necessary. It is essential to keep the wound pockets pus free. Hemorrhage must be guarded against. I have been very fortunate in having very little trouble

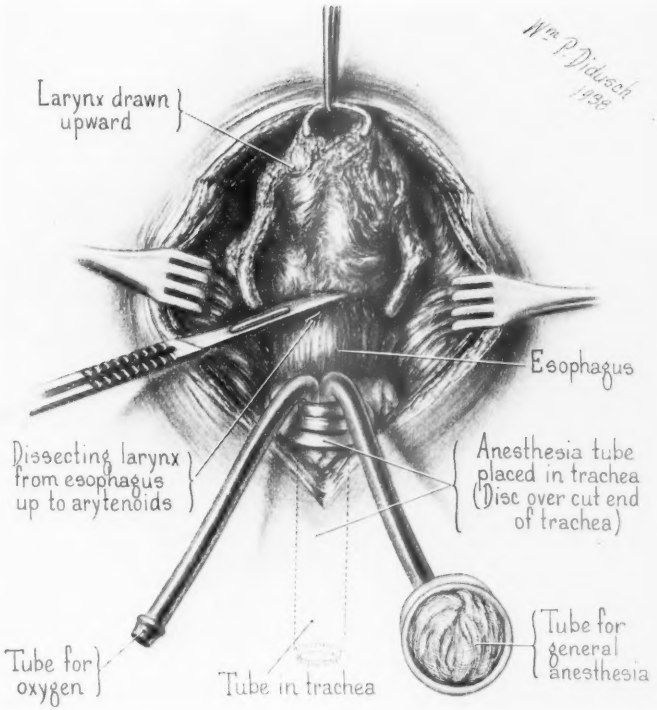

Fig. 7. Dissecting larynx from esophagus.

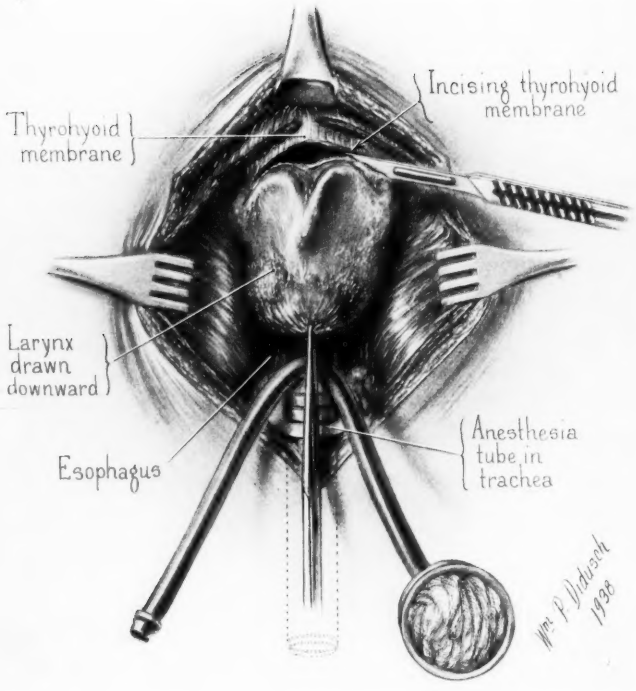

FIG. 3. Incision of thyrohyoid membrane. The larynx drawn downward.

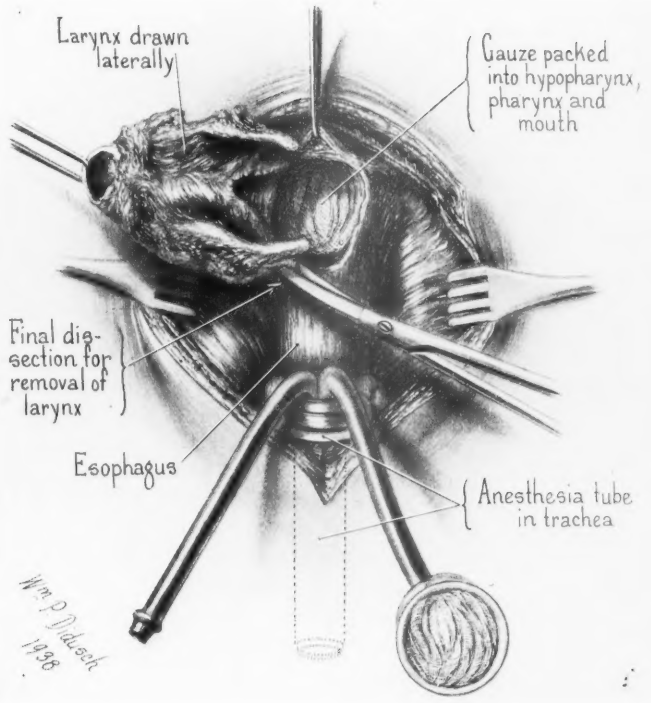

Fig. 9. Final dissection for removal of larynx.

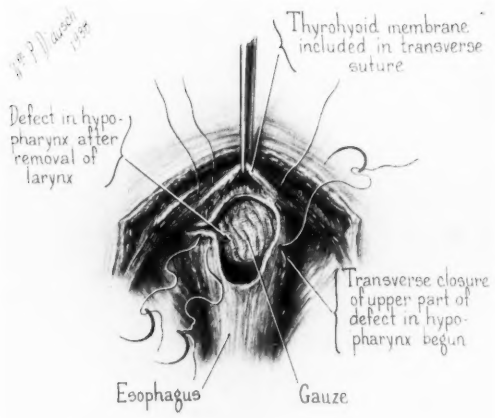

Fig. 10. Transverse closing of the defect in hypopharynx begun.

in this direction, but when it does occur it must be controlled by locating the bleeding point and ligating at once. If it occurs early in the convalescence, it is usually the result of carelessness at the time of operation; if later, it may be due to infection and excessive sloughing. It may be necessary to remove from the trachea dried secretions which collect because the patient can no longer cough them up. This can be done with or without the aid of the bronchoscope, depending on the location of the mass of offending material. Keeping the trachea and bronchi free will help to ward off any tendencies toward bronchial infections and pneumonia. Another complication I had in one case is pharyngeal fistula. This patient had been treated previously with x-ray and radium. As Imperatori reports in a recent article, the closure of these fistulæ in such patients is made more difficult than closure in patients who have had no roentgen therapy.[3]

The feeding tube is left in place about eight days. The usual postoperative procedure as to diet is followed with the exception of the fact that while the tube is being used the food must be in soft or semi-liquid form so as to pass through the tube readily. About the fourth day the patient is encouraged to sit up in bed.

The problem of speech re-education is one which would not, ordinarily, come under the supervision of the surgeon. However,

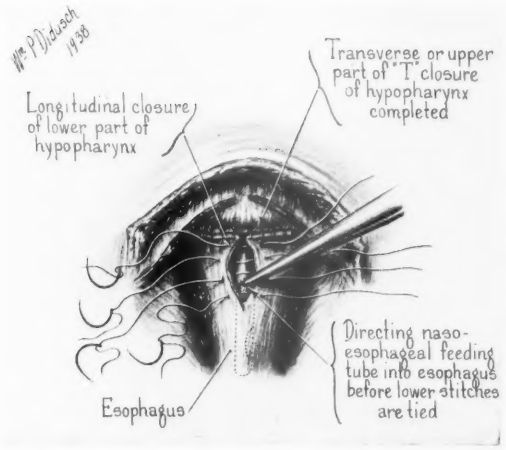

Fig. 11. Directing tube into esophagus before tying stitches.

following total laryngectomy, a not unimportant factor in the cure of carcinoma of the larynx is the production of an artificial or pseudo-voice of some type to rehabilitate the patient to his normal social and economic status. These patients can and have been taught to speak by means of esophageal speech. There is no reason why any patient cannot learn esophageal speech to such a degree that he can be heard and understood in an average sized room and even over the telephone. The use of the artificial larynx is easily mastered but produces an unnatural, monotonous tone the use of which often is quite embarrassing to the patient. For this reason, as soon as the patient is able to swallow, he is trained in esophageal speech. The first teaching periods are confined to demonstration of the reconstructed anatomy of the neck, and from here we proceed to the method of formation of sound. In a paper to be published in the near future, the methods and results of early training in esophageal speech will be discussed in further detail. The pseudo-voice produced depends almost entirely on correct and early training. Several of the latter patients have been able to speak, in a short time, in an audible tone over the telephone. On the other hand, those who had learned to speak by themselves in a whispered voice with a closed esophagus found that it took them as long as several years to overcome this unsatisfactory type of pseudo-voice. Surely, then, in order that we may consider total laryngec-

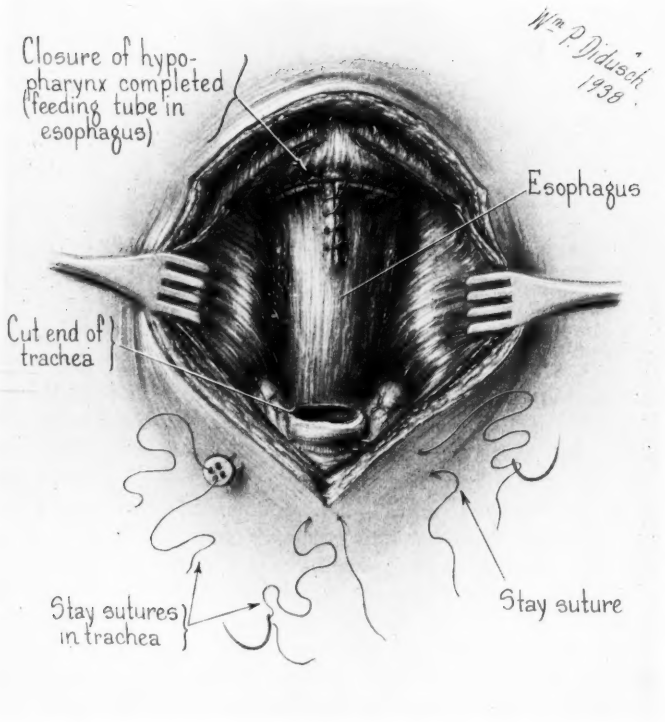

Fig. 12. Closure of hypopharynx completed.

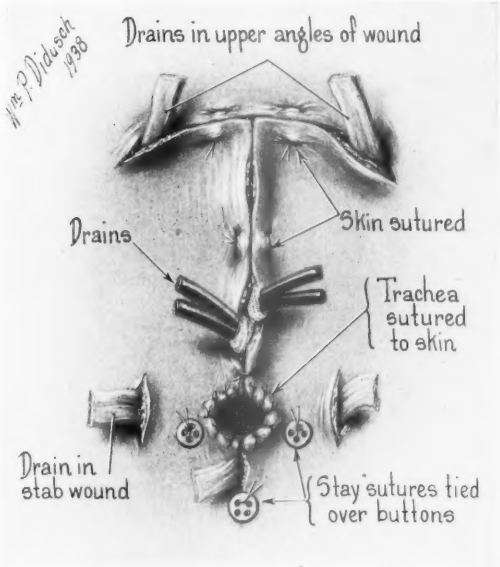

Fig. 13. The trachea sutured to skin. Drains placed and stitches sutured.

tomy a cure for carcinoma of the larynx, it is necessary that, following operation, we instruct, or obtain instruction for these patients in the use of an esophageal voice.

The following chart summarizes the last 25 cases seen in the Bronchoscopic Clinic at Mercy Hospital, Baltimore, and selected as favorable for total laryngectomy. A much larger number of cases was beyond the operative stage and these were treated by palliative measures which merely tend to prolong life and somewhat allay suffering. They are a pitiful example of delayed diagnosis—delayed until it is too late.

A glance at this data serves to emphasize certain points. The mean age of these patients is 51.5 years. The range in years is from 27 to 67. The cardinal symptom in all 25 cases was hoarseness. Seven complained of dyspnea. Two cases were wearing a tracheotomy tube when they presented themselves for examination and

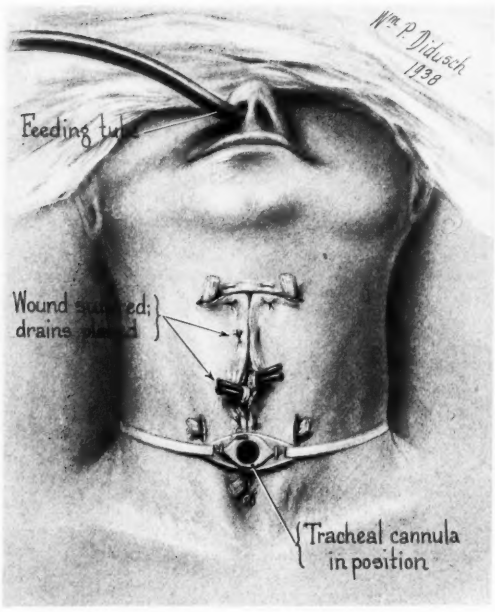

Fig. 14. Operation complete. Canula and feeding tube in place.

complained of marked dyspnea. A third case had had a laryngofissure done four years previously and also complained of dyspnea. Hoarseness had been present as a symptom for periods varying from four months to eight years. Fifteen cases reported loss of weight, many in excessive amounts. Examination of the larynges revealed the lesions to have invaded the larynx at the anterior commissure, or the anterior or middle third of either cord, in one or two cases extending downward to the arytenoid. In the two cases of extrinsic carcinoma included in the series, the glands were distinctly palpable. Six cases had definite fixation of the cords and several others presented partial fixation. The pathological report was squamous cell carcinoma in all but three. One was chondroma, one bronchiogenic carcinoma, and one was unclassified. The results of the series show that of the 25 cases, two survived the operation but died, respectively, on the fourth day (of delirium tremens), and the eighteenth day (no autopsy—cause unknown); four died of recurrence within periods varying from two months to nine months; four died of other

SUMMARY OF OPERABLE CASES

Case	Names	Age	Symptoms	Examination	Pathological Report	Result
1.	J. H. male	58	Hoarseness—loss of weight	Small growth anter. com. and on left cord. No fix. Laryngofis., 6 months before	Squamous cell ca. Intrinsic	Satisfactory recovery. Died 8 years later, other causes—no recurrence
2.	J. O. male	53	Hoarseness, loss weight, cough	Nodular mass mid third right cord. Part. fix.	Squamous cell ca. Intrinsic	Good recovery—patient alive and well today
3.	F. T. male	65	Great loss weight, marked dyspnea trach. tube, 3 months	Large mass filled the larynx. Glands palpable	Bronchiogenic ca. Extrinsic	Good recovery from op. Died 7 months later. Recurrence in neck
4.	V. D. male	48	Hoarseness, 1 year, ch. alcoholism	Mass at ant. 2/3 of both cords and downward	Squamous cell ca. Intrinsic	Survived operation well. Died delirium tremens 4th day
5.	M. K. male	64	Aphonia, trach. tube, 3 weeks, loss weight	Nodular mass filled larynx. Inflam. around tube	Squamous cell ca. Intrinsic	Recovery was delayed—ultimately good. Died 1 year later—cause ? ?
6.	C. H. male	50	Hoarseness, 8 years, intermittent	Mass on right cord and downward	Squamous cell ca. Intrinsic	Patient made good recovery. Living and well
7.	M. I. female	60	Hoarseness, 6 months, dyspnea	Mass on ant. 2/3 right cord	Squamous cell ca. Intrinsic	Recovery uneventful for 2 weeks. Died suddenly 18th day. No autopsy
8.	S. R. male	55	Hoarseness, 9 months, loss weight	Growth mid-third left cord. Some fixation	Squamous cell ca. Intrinsic	Recovery good. Gained 30 pounds weight. Died 6 years later—fractured skull

SUMMARY OF OPERABLE CASES—(Continued)

Case	Names	Age	Symptoms	Examination	Pathological Report	Result
9.	J. H. male	51	Hoarseness, 1 year, loss weight, dyspnea	Thyrotomy revealed large mass left side	Chondroma	Good recovery. Living and well today
10.	F. G. male	27	Hoarseness, cough, loss weight	Mass involving entire left cord and down to first tracheal ring	Squamous cell ca. Intrinsic	Rapid recovery. Patient living and well
11.	J. H. male	65	Hoarseness, 18 months, loss weight	Growth mid-third left cord with fixation	Squamous cell ca. Intrinsic	Recovery good. Sudden death 4 years later—heart
12.	M. T. female	52	Hoarseness, 9 months	Nodular growth left cord and ant. 2/3	Squamous cell ca. Intrinsic	Good recovery. Living and well
13.	J. S. male	50	Hoarseness, 1 year, ch. cough, loss weight	Growth involving cord and ant. commissure	Squamous cell ca. Intrinsic	Living and well. Rapid recovery
14.	J. G. male	46	Hoarseness, 8 months, loss weight, dyspnea	Growth on left cord. Fixation present	Squamous cell ca. Intrinsic	Recovery good but died 2 years later—metas. rib
15.	W. E. male	48	Hoarseness, 2 years, loss weight	Large mass on left cord and downward. Fixation	Squamous cell ca. Intrinsic	Recovery good. Living and well
16.	M. H. male	48	Hoarseness, 8 months, loss weight and pain, ch. alcoholism	Growth on left cord ext. to arytenoid. Glands pal.	Squamous cell ca. Extrinsic	Recovered and returned to work. Died suddenly 9 months later

17.	C. K. male	67	Hoarseness, 6 months, loss weight and pain, ch. bronchiectasis	Extensive growth left cord with fixation	Squamous cell ca. Intrinsic	Rapid recovery. Developed abscess base right lung. Responded to bron. drain
18.	S. W. male	49	Dyspnea and loss weight, aphonia	Laryng. apert. closed by recur. growth following radium after laryngofis. four years previously	Squamous cell ca. Intrinsic	Recovery rapid. Difficulty with final closure due to x-ray therapy. Living and well
19.	H. C. male	48	Hoarseness, 4 months, loss weight	Small growth ant. com. invol. ant. third right cord. Fixation	Squamous cell ca. Intrinsic	Quick recovery. Patient living and well. Good esophageal voice
20.	H. O. male	63	Hoarseness, 6 months	Nodular growth ant. 2/3 left cord and ant. third right cord	Squamous cell ca. Intrinsic	Good recovery. Living and well
21.	L. M. female	62	Hoarseness, 8 months	Nodular growth ant. 2/3 left cord. Contact ulcer on right cord	Squamous cell ca. Intrinsic	Good recovery. Living and well
22.	J. S. male	28	Hoarseness, several months	Mass at anterior com. thickening at ant. ends both cords	Squamous cell ca. Intrinsic	Patient living and well. Rapid recovery
23.	R. P. male	47	Hoarseness	Growth on the left cord	Squamous cell ca. Intrinsic	Good recovery. Living and well
24.	R. S. male	63	Hoarseness, several months	Mass on right cord	Squamous cell ca. Intrinsic	Speedy recovery. Living and well
25.	G. G. male	56	Hoarseness and dyspnea	Growth involved left cord and extended down into trachea	Unclassified	Survived operation and lived two months. Died recurrence

causes within periods varying from one year to eight; and fifteen are living and well at the present time. The postoperative period of those living varies from one year to nine.

SUMMARY

A series of 25 cases of carcinoma of the larynx on whom total laryngectomy was performed is presented. The symptoms of the disease pointing to proper and early diagnosis are pointed out. The technique of the one-stage operation is given in detail with complete illustrations. After-treatment and possible complications—hemorrhage, pharyngeal fistula and bronchial infections are discussed. The fertile field of speech re-education following this operation was indicated as the means of securing for the patient social, mental and physical well-being for the remainder of his life.

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XXXVIII

THE PARENTERAL ADMINISTRATION OF CERTAIN SUBSTANCES IN UPPER RESPIRATORY INFECTIONS*

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WARREN B. DAVIS, M.D.
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PHILADELPHIA

L. W. Dean[1] recently made the following statement: "Laryngology is making great progress because laryngologists are internists with special laryngologic training. We not only consider the influence of laryngologic conditions upon the organism as a whole, but we are particularly concerned with the influence of systemic disturbances upon the nose and throat. The latter is doing much for our patients."

In the motley group of nasal dysfunction cases that confront the laryngologist daily in his office, many etiologic factors must be looked for and considered. Some of these are as follows: poor hygiene, dietary deficiencies and excesses, metabolic disturbances, improper clothing, climatic conditions and changes, including lack of sunshine, lowered or excessive humidity and faulty ventilation in the homes, allergy in one or more systems, neuro-endocrine imbalances, faults in structural hygiene, septic foci in the tonsils, sinuses and/or other loci, structural faults in the nose, causing blockage, parenchymatous changes in certain viscera (nephrosis), infections of the upper respiratory system secondary to exanthemata or other contagious diseases, lack of normal vascular tone, fatigue, faulty elimination, exogenous and endogenous poisons, subnormal oxidation, swimming or prolonged chilling in the water, psychogenic conditions, organic or functional, either hereditary or acquired or both, other stresses and strains produced by the present speed of living, effecting small grades of exhaustion of the kinetic system (brain, adrenals, thyroid. muscles and liver) resulting in diminished vascular tonicity of the body in general, and of the mucosa of the upper respiratory system in particular, ad infinitum.

*Read before the Section on Otolaryngology of the College of Physicians of Philadelphia, January 19, 1938.

It is obviously impossible for the otolaryngologist to attend to all of these etiological factors. Infected tonsils and adenoid masses are removed. Structural faults in the nose are corrected with the view to putting this organ in the best possible mechanical state. Time-proved conservative measures are used to institute optimum aeration and drainage. They consist, in the main, of shrinking sprays, packs, infra-red irradiation, lavage, displacement methods, topical application of appropriate medicaments, judicious use of negative pressure, postural drainage, removal of polypi, etc. Where necessary, suitable stock vaccines, three unit doses of insulin, Lugol's solution in three drop doses, nonspecific protein therapy and similar agents, are added for the purpose of heightening the defensive mechanisms and promoting metabolism. In all cases we attach a great deal of importance to personal hygiene and corrective diet, for it is patent that, other things being equal, the greater the bodily vigor, the quicker will the "restitutio ad integrum" be effected.

Some cases may require the co-operation of the internist for further study, to the end that better results be obtained. All patients giving an allergic history, or presenting symptoms thereof, are referred to the allergist for his specialized help.

In spite of our best efforts and those of our consultants, we are often disappointed in our results. In general, however, we can say that satisfaction has resulted in the majority of our cases.

Our most refractory cases have consisted of those exhibiting fair to good intranasal mechanics with mucosal intumescence and excessive postnasal secretions. Though they were the most difficult to relieve, a certain proportion were benefited by the oral administration of calcium, strychnine sulphate, thyroid extract, atropine sulphate, dilute hydrochloric acid, bicarbonate of soda, sodium chloride, phenobarbital, ephedrine sulphate and similar agents.

This led us to consider further avenues of approach, for we found that the oral administration of the above medicaments along with our office treatment did not give us all the results that we wished.

It has been our feeling for a long time that parenteral administration of various substances might be of further aid to us in the more refractory cases. Endocrine agents were our first thought, but this was abandoned because at the present time the relationship between endocrinology and otolaryngology is still "terra incognita." We considered the use of simpler medicaments, and reviewed briefly

the authoritative works. The substances that we chose were the following: camphor, menthol, iodoform, iodides in an oil vehicle.

Inasmuch as this paper is confined to the parenteral administration of these substances, we shall omit most of the references to the physical and chemical properties thereof. Such details can be obtained in any standard work on pharmacology and materia medica, in addition to those about to be quoted.

Horatio C. Wood[2] states that the action of camphor stimulates the respiratory center and probably the heart muscle and that it widens the blood vessels due to central and peripheral influences. Though it is of distinct value in adynamic conditions and sudden cardiac failure when given hypodermically, he makes the point that it should not be relied upon alone, to the exclusion of more potent drugs. Solis-Cohen and Githens[3] opine that though much controversy exists regarding the action of camphor on the circulation, because of great divergence of opinion, it is useful in catarrhal affections of the respiratory tract, especially bronchitis. In reference to menthol, they[4] tell us that its action on the heart and vessels resembles camphor, the heart being stimulated by very small doses of menthol. They[5] stress the point that iodides increase the destructive or catabolic side of metabolism of all cells, especially the diseased cells. The above mentioned authors[6] likewise give us a vivid account of how J. Solis-Cohen applied an emulsion of iodoform in olive oil or glycerine, placing it on a brush or sponge and firmly holding it over the back of the throat while the patient swallows, so that the arytenoid eminences and associated structures are coated with it. They[7] then state the following: Iodoform dissolved in olive oil or almond oil alone or with terebine and eucalyptol, 5 per cent in liquid vaseline, injected hypodermically or into the muscles, is of benefit in various forms of pulmonary infections; not only in obstinate bronchitis, acute or chronic, or even bronchopneumonia, but also in pulmonary tuberculosis accompanied by fever, whether or not cavitation or mixed infection can be demonstrated."

Hare[8] felt that camphor given internally is beneficial in the coryzas, though he makes no mention of any parenteral use of this drug. Menthol he[9] values for inhalation purposes, as well as the tincture of iodine. He[10] also extols the virtue of the latter. He[11] makes the statement that iodoform is of value when used internally, in tertiary syphilis; in a powder blower in the hoarseness and discomfort of laryngeal phthisis, or as a spray by using two grains of iodoform in equal parts of spirits of turpentine and sweet oil. He ends with the statement that several clinicians have obtained good results

in the early stages of phthisis by daily hypodermic injection into the back of 30 minims of a 1-100 solution of iodoform in oil. Bastedo[12] says that camphor is employed as a circulatory stimulant hypodermically in alcohol, ether or oil, and that these solutions are irritants. Cushny[13] remarks that the iodides are appropriate for expectorant purposes to render the bronchial mucus more watery and less tenacious and thus to facilitate its removal. It is of value in some cases of asthma. The same author[14] goes on to say that iodoform is readily decomposed in the presence of alkaline fluids and in protein solutions, liberating iodine. After iodoform has been absorbed, iodine has been shown to be present in the saliva, perspiration and bronchial secretions. It is excreted in the urine in the form of iodides. The tissues apparently retain it very tenaciously, for iodides have been found in the urine for more than a month after the administration of iodoform.

Sollmann[15] tells us that the parenteral administration of camphor has some stimulating effects on the circulation, improving the volume, tension and regularity of the pulse and that the weakened heart muscle may respond better to camphor than animal experimentation would indicate. The stimulation may be reflex, since the injections produce considerable local reaction. Concerning iodoform, he[16] states that it acts by liberating iodine, since by contact with tissues or their extracts or bacteria, this phenomenon slowly takes place. He states further that Hamburger found that it stimulates phagocytosis, and that according to W. Weil (1913), the injection of iodoform leads to a persistent leucocytosis in dogs.

It seemed evident, therefore, that persistent and systemic use of the above enumerated substances might aid us in relieving some of the more refractory cases. We felt it necessary to combine these substances together for the purpose of gaining every likely value that the action of these drugs might bring out, especially as regards the circulation and the metabolism of cells. The favorable comments of the authorities quoted above made us feel that we were not relying wholly on empirical reasoning.

Our next step was to select a preparation which would meet the requirements.

Specifically, our attention to the parenteral administration of the substances mentioned above was caught by continued reports of the success of a certain group who have, for a number of years, made use of this method for the treatment of hayfever, asthma and sinus infection and, as far as could be ascertained, of these drugs, although their exact formula was secret. In searching for a stand-

ardized commercial product that would fulfill our requirements, we tried at least one other, without much success, but recently have used the combination called Camirol, the formula of which, certified by the manufacturers, consists of 2 per cent camphor, 4 per cent menthol, 1 per cent iodoform, 0.1 antimony iodide in peanut oil. Its advantage is that it is put up in sterile ampules, in accurately measured dosage, so that its administration is easy. In the majority of cases so treated the injection has been given intramuscularly, but from more recent experiments, it seems likely that the subcutaneous route is just as efficacious. It has been advised by the makers of this preparation, and as practiced by the group mentioned above, that the contents of one ampule, 1 cc., be injected daily for four days and thereafter semi-weekly until definite improvement has taken place, and then at increasingly longer intervals. This course has admittedly not been followed in this series, the injections having been made at much wider intervals to suit the patients' convenience. Where one or two doses only are recorded in Chart I, there were two reasons: (1) because the patients recovered sufficiently so that a second did not seem to be indicated, and (2) because the patients did not return at an interval sufficiently short to seem to make further treatment by this method worthwhile. Obviously, the cases that showed marked improvement after one or two doses were the acute cases, while those who failed to show early improvement belonged mostly in the chronic classification.

It is freely admitted that this is not a scientific study, as our classification of cases is a loose one, comprising acute, subacute and chronic cases without distinction as to the type of sinus involvement and with some allergic or vasomotor cases included.

Our impression is that cases showing evidence of an endocrine imbalance were more responsive to this form of therapy. Also, since all the individuals reported upon were private patients, not human guinea pigs, who came to us for relief from urgent symptoms, ordinary routine methods of treatment were given in most instances, so that a true evaluation of the merit of this method is not possible under these circumstances. Nevertheless, patients who had previously resisted, or showed no favorable results from these routine treatments, did, it seemed to us, in many instances, improve markedly when the injections were added. The injections are practically painless, there is no reaction and in only a very few were there any unpleasant after effects, such as induration at the site of injection.

As stated before, the vehicle used in this preparation is peanut oil. This has a twofold value; it is highly bland and it is less apt

CHART I

Two hundred and eighty-four patients were given 1,101 injections of camphor menthol-iodoform solution (Camirol) from April 9, 1937, to January 15, 1938.

Number of Patients	Number of Injections	Result A	Result B	Result C	Result D
72	1	4	8	56	4
52	2	12	15	24	1
36	3	17	12	7	—
44	4	23	12	9	—
19	5	5	10	4	—
12	6	3	7	2	—
7	7	4	2	1	—
14	8	9	5	—	—
9	9	3	6	—	—
4	10	2	2	—	—
5	11	3	2	—	—
5	12	2	2	1	—
1	13	1	—	—	—
1	14	1	—	—	—
1	15	—	1	—	—
1	16	—	—	1	—
1	19	1	—	—	—
284		90 (32%)	84 (30%)	105 (36%)	5 (2%)

to become rancid than most other oils. Moreover oil, as a vehicle, is slowly absorbed, resulting in a slower and more prolonged action of the aforementioned chemicals.

The preparation can be drawn up into the syringe much more easily if the ampule is placed on the sterilizer tray and warmed for a minute or two. This procedure thins the oil vehicle. A No. 22 gauge needle will be found appropriate.

COMMENT

We wish to emphasize again that this presentation is not a scientific study, for the results reported have not been tested in the crucible of the laboratory, but represent our critical evaluation of each individual private patient based upon subjective, objective and meticulous intranasal examination. This is a preliminary report only on a method which, from the impression gained from a study of our results, we think merits further trial, at least as an adjunct to approved and tested procedure. It will not supercede surgery, where surgery is definitely indicated, but even here it may well contribute to the eventual success of surgical procedures.

Our most striking results were obtained in the acute cases and in the exacerbations of low-grade chronic sinusitis, though some chronic cases exhibiting minor grades of endocrine imbalance were benefited.

SUMMARY

1. Two hundred and eighty-four cases comprising acute, subacute and chronic forms of sinusitis without distinction as to type, including allergic and endocrine disturbances, are presented.

2. They had previously resisted or shown no favorable results from the routine treatments.

3. They were given parenteral administration of camphor-menthol-iodoform solution (Camirol) at intervals, in addition to the aforementioned routine procedures.

4. The intervals of injection were three or four days in the acute types, and where longer injection therapy was indicated, they were lengthened to one or two weeks.

5. Very gratifying results were obtained in 32 per cent of the cases (column A); beneficial results in 30 per cent (column B); uncertain findings in 36 per cent (column C); unsatisfactory results in 2 per cent (column D).

CONCLUSIONS

The percentage of favorable results leads us to believe and conclude that this preparation merits further use and study. It is not a cure-all, but has definite pharmacodynamic value. It should be used as an adjunct to time-proved and precise office measures in the various types of acute and chronic sinusitis, catarrhal or suppurative.

1721 PINE STREET.

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XXXIX

MODERN CONCEPTS OF LARYNGEAL TUBERCULOSIS*

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LIBERTY, N. Y.

The purpose of this paper is to present some of the modern concepts of laryngeal tuberculosis. This discussion is based on clinical observation and experience with laryngo-pulmonary tuberculosis over a period of twenty-five years, both in institutions and in private practice.

Before proceeding with the subject of laryngeal tuberculosis I feel that it is pertinent to discuss briefly the present day views on the pathogenesis of pulmonary tuberculosis. Although there is still much to be proved, it is generally conceded that, following the primary infection of the lung and conjoined tracheo-bronchial lymph nodes during early life (primary complex), invasion of the lungs by the tubercle bacillus either from within, (endogenous) or from without, (exogenous), is caused later in adult life by way of the blood stream (hematogenous) or through the air passages (broncho-pulmonary, also spoken of as bronchogenic). Accordingly, for all practical purposes pulmonary tuberculosis in this discussion will be spoken of as hematogenous or broncho-pulmonary. Here it may be well to add that although pulmonary tuberculosis may exist as hematogenous or bronchogenic during its early manifestation, after the pathologic process is well developed it usually takes on both forms; that is, a hematogenous lesion may ulcerate through the alveoli and become bronchogenic, while a bronchogenic lesion may break into the circulation and take on a hematogenous course. So, therefore, in speaking of hematogenous or bronchogenic, what is usually meant is predominantly hematogenous or predominantly bronchogenic. Hematogenous tuberculosis may run from the very severe and usually fatal generalized miliary tuberculosis to the very benign and localized tuberculosis, which may appear in discrete and limited areas of the lungs, eventually clearing up without leaving a trace. Broncho-pulmonary tuberculosis, in this discussion, will

*Read before the Section of Otology, Rhinology and Larngology of the Pan-American Medical Association, January 21, 1938, at Havana, Cuba, and the Tuberculosis Sanatorium Conference of Metropolitan New York at Cornell University Medical College Amphitheatre, New York City, N. Y., February 9, 1938.

embrace all forms of chronic pulmonary tuberculosis including exudative, caseous, fibrocavernous and all the productive forms. According to Miller[1] about twenty to thirty per cent of all cases of pulmonary tuberculosis may be reasonably ascribed to hematogenous dissemination, while Ornstein and Ulmar[2] believe that not more than fifteen to twenty per cent at the very most of adult tuberculosis is the direct manifestation of hematogenous tuberculosis.

ETIOLOGY

Tuberculosis of the larynx is always secondary to a tuberculous infection elsewhere in the body and usually to that of pulmonary tuberculosis. In a certain number of cases of hematogenous tuberculosis the infection of the larynx probably occurs simultaneously with the infection of the lungs. The author has never seen a case of primary tuberculosis of the larynx, authough theoretically, such an infection could occur. The few cases of primary tuberculosis of the larynx which are reported in medical literature were mostly in the pre-x-ray era and were probably found in that type of case of hematogenous tuberculosis where physical signs in the chest are definitely lacking. With our present methods of diagnosis, and especially the x-ray, I feel that the original pulmonary lesion could have been detected in most of these cases. It has also been the author's observation that laryngeal tuberculosis occurs only in active cases of pulmonary tuberculosis. Once the pulmonary lesion becomes healed the larynx remains uninvolved.

MODE OF INFECTION

The mode of infection is most important and to my mind constitutes the crux of the entire problem. Once it is ascertained, it becomes relatively easy to render a fairly accurate prognosis and the management of the case can then be placed on a more scientific and practical basis, thereby becoming more efficient.

There are probably three modes of infection by which the larynx becomes involved:

1. Surface infection, through the laryngeal mucosa, which is undoubtedly the most common of all.

2. Hematogenous route, which is rather uncommon generally, but very preponderant in cases of hematogenous tuberculosis.

3. Lymphogenic route, which probably exists, but cannot definitely be proved.

1. *Surface Infection:* The theory of surface infection of the larynx is the most plausible and is probably true of the vast majority

of broncho-pulmonary cases. In 1914, when I was resident physician at the Otisville Sanatorium, I made a survey of laryngeal tuberculosis among the 500 adult patients at the Sanatorium. I found a positive sputum in 91% of the laryngeal cases. I made a similar study at the same Sanatorium in 1934, examining a similar group of 500 patients and found that the sputum was positive in 98% of the cases. The almost constant presence of a positive sputum in these cases, makes the surface infection theory very convincing and especially when we know that tubercle bacilli can penetrate the mucous membrance of the larynx even when it is in a healthy state. In tuberculous patients, however, the mucous membrane of the larynx is generally traumatized and inflamed which makes the passage of tubercle bacilli to the subepithelial structures so much easier. Because of the above findings and with the exception of some early hematogenous cases, I have always felt and still do that it is not safe to make a diagnosis of tuberculous laryngitis unless we have a positive sputum.

2. *The Hematogenous Route:* This, I believe, is rare in broncho-pulmonary tuberculosis but very common in hematogenous pulmonary tuberculosis. This type of laryngeal infection I described in 1916[3] and 1922[4] under the peracute and acute types. In these particular types I believe that the larynx becomes involved simultaneously with the lungs. The laryngeal lesion is usually severe and overshadows the pulmonary lesion and, for this reason, these patients generally come to the doctor because of symptoms referable to the larynx and the diagnosis of the pulmonary condition is often made afterwards. A physical examination of the chest will generally reveal no significant findings and one may even fail to find the pulmonary lesion on fluoroscopy, but on x-ray examination the pulmonary lesion will probably be revealed as that of a fine peppering of the parenchyma, which may be disseminated through one and often both lungs, characteristic of generalized miliary tuberculosis. This particular kind of laryngeal lesion may also occur in the less severe forms of hematogenous pulmonary tuberculosis characterized by disseminated miliary nodules with little or no general evidence of generalized miliary tuberculosis. The sputum in these cases may often be found negative until ulceration occurs either in the lungs or the larynx and then it will be positive. In these cases I feel that there is no question that the larynx becomes involved by way of the blood stream.

3. *The Lymphogenic Route* of laryngeal infection probably exists but is difficult to prove. In discussing the etiology of laryn-

geal tuberculosis Wood[5] states "Certain authorities place importance upon the possibility of a lymphatic route, but if this ever takes place, it is an extremely rare happening, as the laryngeal lymphatics are well isolated from the surrounding structures. It is just within the limits of possibility that in cervical tuberculous adenopathy there might be a retrograde lymph current which would carry the bacilli up the efferent lymphatics of the larynx." Cases have been reported where a laryngeal lesion occurred secondary to involvement of the tonsils, cervical lymph nodes and bronchial glands. I can recall several cases where the larynx was involved in the presence of extensive tuberculosis of the cervical glands; also a number of cases of tuberculosis of the larynx following a tonsillectomy. In the latter instances, however, I was never certain whether the laryngeal lesion existed to a certain extent before the tonsils were removed or perhaps the larynx became involved afterward. Once the larynx has become invaded it is quite probable that the spread to the other portions of the larynx is by way of the lymphatics[6].

CLINICAL TYPES

From many years of observation I feel that laryngeal tuberculosis can be conveniently classified into four clinical types—peracute, acute, subacute, chronic. The peracute and acute laryngeal lesions undoubtedly occur through the blood stream while the subacute and chronic lesions most probably occur through surface infection.

Peracute, or miliary tuberculosis of the larynx, is characterized by very severe symptoms referable to the larynx. Dysphagia is often the first symptom to appear. This particular type is most often found in generalized miliary tuberculosis and also in the fulminating hematogenous cases. Laryngoscopic examination reveals severe swelling of the epiglottis and perhaps all the other structures of the larynx. Macroscopic tubercles can often be seen. The arytenoids are usually involved and the swelling extends to the epiglottidean folds. There is a rapid tendency to ulceration and necrosis and in addition to the dysphagia the patient also has dysphonia when the deeper structures of the larynx become involved. The patient often complains of sharp pain radiating to one or both ears. Perichondritis is generally present. The pharynx and especially the anterior and posterior pillars, tonsils and sometimes the uvula become involved. The patient runs a high fever in the afternoon, about 102 to 104 degrees, and he usually succumbs in from three to eight weeks from inanition and the overwhelming toxemia, often terminating in meningitis. Treatment is of no avail and such a lesion

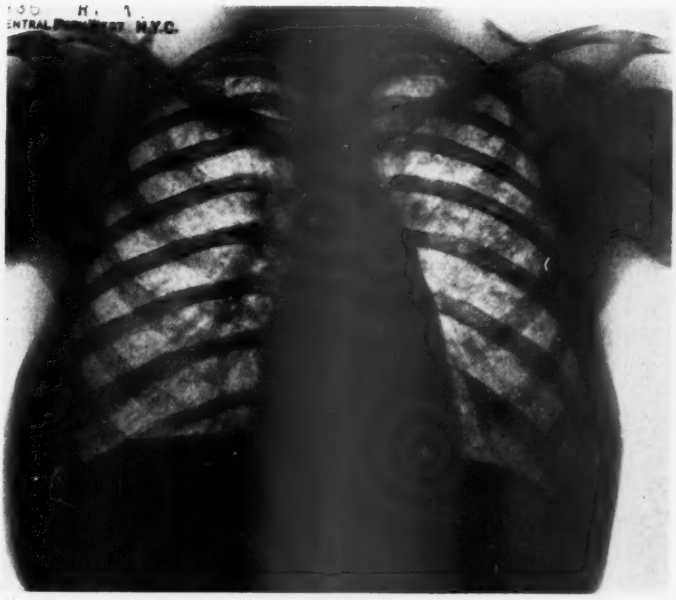

Fig. 1. Case 1. B. R. Acute generalized tuberculosis complicated by peracute tuberculosis of the larynx and pharynx with involvement of the tonsils, uvula and cervical glands. (See Figs. 2 and 3.) Died of tuberculous meningitis six months after admission.

in this particular type of pulmonary tuberculosis can still be labeled as a "Death Warrant."

PATHOGENESIS OF LARYNGEAL TUBERCULOSIS

PULMONARY TUBERCULOSIS

Hematogenous 25%	*Broncho-pulmonary 75%*
Laryngeal tuberculosis	Laryngeal tuberculosis
Mostly blood stream infection	Mostly surface infection
Occurs frequently, 80%	Less frequent, 14.6%
Mostly peracute and acute	Mostly subacute and chronic and occasonally acute

REPORT OF CASE

Case 1.—B. R., female, age 22. Admitted January 13, 1937. Referred by Dr. George G. Ornstein. Both family history and previous history negative.

Fig. 2.

Fig. 3.

Case 1. Peracute tuberculosis of the larynx and pharynx.

Physical examination showed that the patient was markedly emaciated. Examination of the chest revealed extensive bilateral dissemination characteristic of hematogenous pulmonary tuberculosis (Fig. 1).

Laryngoscopic examination showed extensive edema with ulceration of the epiglottis, also both arytenoids with extension into the epiglottidean folds. Both vocal cords were infiltrated and ulcerated (Fig. 2). The pharynx also was extensively involved, including both anterior and posterior pillars, tonsils and uvula (Fig. 3). Patient continued a downhill course. Condition of both larynx and pharynx progressed rapidly and dysphagia became extremely pronounced, so that the patient was unable to take nourishment. The superior laryngeal nerves were injected with alcohol for the relief of the dysphagia. Patient finally developed terminal meningitis and died July 13, 1937.

Diagnosis: Peracute tuberculosis of the larynx, pharynx and tonsils.

Summary: The above illustrates a fairly typical case of peracute laryngeal involvement, complicating an acute generalized miliary tuberculosis.

The acute clinical type is mostly seen in hematogenous pulmonary tuberculosis and occasionally in broncho-pulmonary tuberculosis, which has become hematogenous sometime during the course of the disease. In this particular type the course is less acute than in the peracute. Here the patient also complains of laryngeal symptoms which are, however, less severe than in the peracute or miliary type. Laryngoscopic examination shows swelling of the epiglottis (sausage shaped) and perhaps one or both of the arytenoids with extension into the epiglottidean folds (pear shaped). Other parts of the

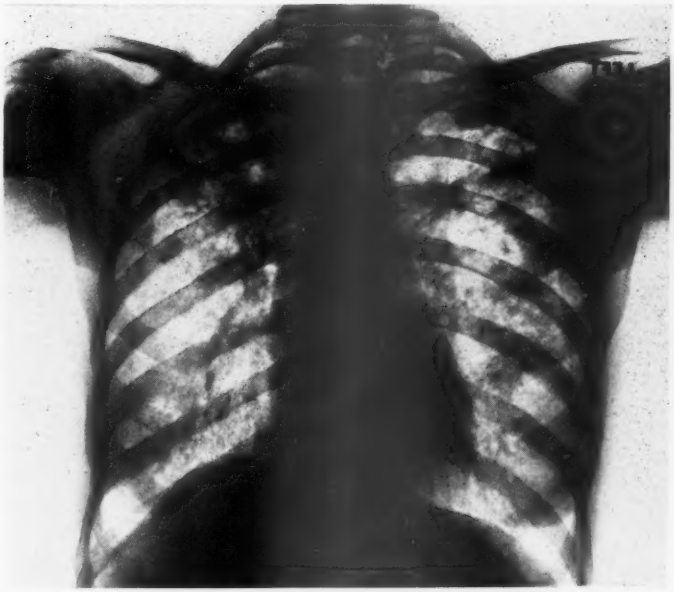

Fig. 4. Case 2. M. C. Pulmonary tuberçulosis, advanced, complicated by acute tuberculosis of the larynx (Fig. 6). Consolidation right upper lobe with probable cavitation, with dissemination of lower portion of right lung and entire left lung, characteristic of hematogenous tuberculosis. Sputum positive.

larynx, such as the ventricular bands and vocal cords, may also be affected. These cases, too, go on to ulceration but often there is a tendency to resolution and to fibrosis and healing, and especially in cases where the pulmonary lesion shows a tendency to retrogression. These laryngeal lesions are amenable to treatment and the course of this type of lesion will usually follow the course of the pulmonary lesion.

REPORT OF CASE

Case 2.—M. C., female, age 27. Referred by Dr. Foster Murray. Admitted to Liberty February 1, 1937. Family history negative. Previous history: right mastoid at ten years of age. Present history dates back one and one-half years, at which time, while, pregnant, patient had a pleurisy on the left side. Following childbirth she developed a cold, sore throat, malaise, fever and lost weight, from 121 to 98 pounds, also persistent cough with expectoration and night sweats. There was hoarseness for one year before she came to Liberty and her sputum was

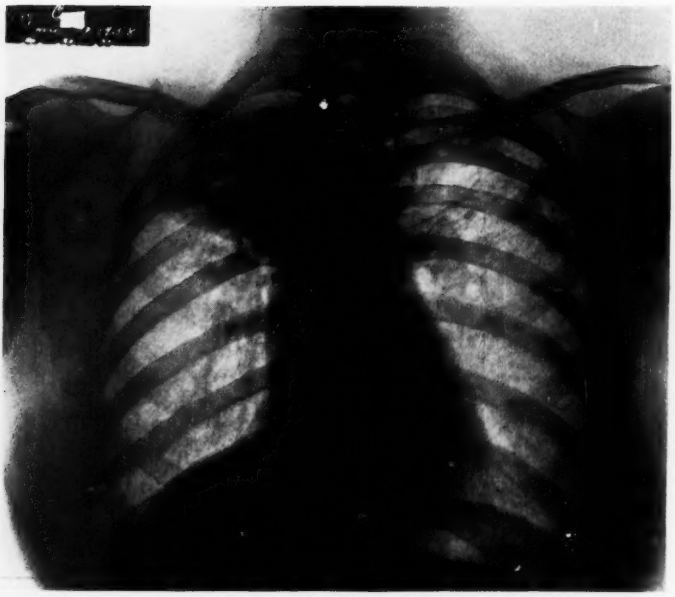

Fig. 5. Case 2. M. C. One year later. Lesion right upper lobe more or less stationary, probably more organized. Marked clearing both middle and lower lobes on the right side and also the entire left lung with evidence of fibrosis at the left upper lobe. Sputum negative. Laryngeal lesion definitely healed (Fig. 7).

positive. When first seen in March, 1937, the patient weighed 100 pounds; temperature from 98 to 100 degrees; pulse 110 to 120; respiration 20 to 22.

Physical examination showed a consolidated area in the right upper lobe with probable excavation, also signs of disseminated lesion in the left upper lobe.

X-ray examination, January 13, 1937, revealed a pneumonic lesion in the right upper lobe with diffuse infiltration extending into the right middle and lower lobes. The left lung field showed a disseminated lesion throughout the left lung, characteristic of hematogenous tuberculosis (Fig. 4).

Laryngoscopic examination showed a moderate edema with ulceration of the left half of the epiglottis (Fig. 6).

Clinical course: Patient was treated conservatively with complete bed rest. She became afebrile and improved generally, having gained forty pounds in weight. Sputum was consistently positive until February 8, 1938, when it was found negative.

Fig. 6.

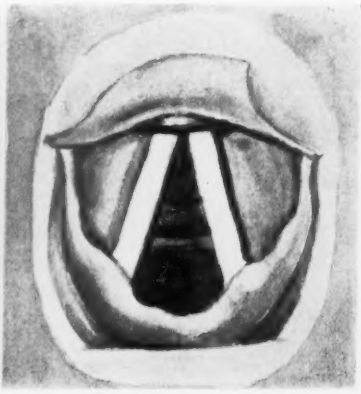

Fig. 7.

Case 2. Acute tuberculosis of the larynx.

X-ray examination of January 31, 1938, reveals a heavy infiltration well limited to the right upper lobe, with possible cavitation. There has been marked clearing of both middle and lower lobes on the right side and also of the entire left lung, with evidence of fibrosis of the left upper lobe (Fig. 5).

The larynx shows marked improvement. Edema of the epiglottis has entirely disappeared and the ulceration of the left portion of the epiglottis is definitely healed (Fig. 7).

Diagnosis: Acute tuberculosis of the larynx complicating a predominantly hematogenous pulmonary lesion.

Summary: This case illustrates an acute laryngeal lesion secondary to hematogenous pulmonary lesion, which healed as the pulmonary lesion improved.

The subacute type is characterized by a pseudo-edema of part or parts of the larynx with a strong tendency to fibrosis. It may begin as such or else follow the acute or chronic type. This clinical form of laryngeal tuberculosis is usually found in the exudative forms of pulmonary tuberculosis and the infection probably occurs in most of the cases by the surface route. Papilliform infiltrates and soft polypoid excrescences usually belong to this type. The local symptoms may be slight or moderate, depending largely on the extent and site of the lesion. Hoarseness as a rule is present particularly when the true vocal cords or the interarytenoid sulcus is involved. Dryness in the larynx is usually complained of, especially when the fibrosis is extensive. With the patient in good general condition it

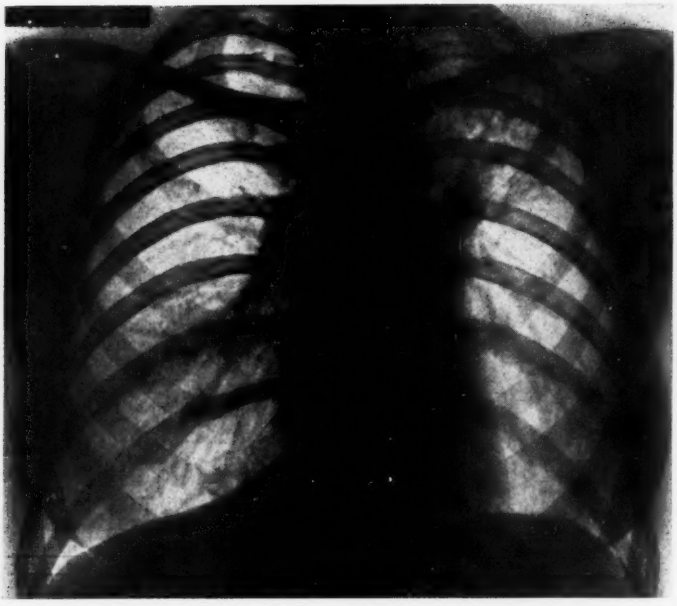

Fig. 8. Case 3. H. LaH. Pulmonary tuberculosis, moderately advanced and active complicated by subacute tuberculosis of the larynx with infiltration of the posterior third of both vocal cords and also posterior commissure (Fig. 10). Consolidation of left upper lobe and small cavitations. Sputum positive.

runs its course and may become completely arrested. The prognosis as to life and voice depends much on the patient's pulmonary lesion and on early diagnosis. With proper treatment the prognosis in the average subacute case is favorable.

The following case illustrates the above type:

REPORT OF CASE

CASE 3.—H. LaH., female, age 29. Referred by Dr. Foster Murray April 19, 1937. Family history negative. Previous history: pleurisy about seven years before admission. Present history dates back to July, 1936, at which time patient had a persistent cold. Examined by Dr. Murray, who made a diagnosis of pulmonary tuberculosis. When first seen on April 19, 1937, patient weighed 121.6 pounds, complained of cough, slight expectoration and hoarseness.

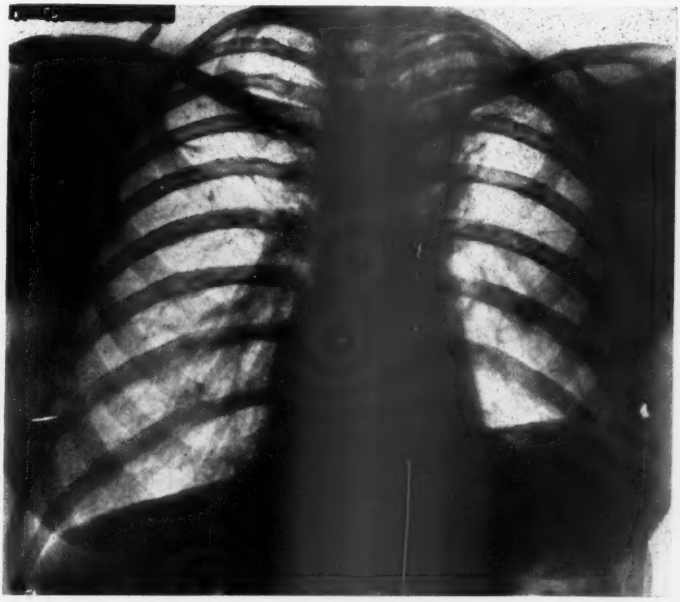

Fig. 9. Case 3. H. LaH. Two months and twenty-four days after left phrenic crush. Definite clearing of the lesion in left upper. Sputum negative. Larynx normal (Fig. 11).

Physical examination revealed signs of consolidation in the left upper lobe with cavitations (Fig. 8). Examination of larynx showed a slight thickening of the posterior commissure and infiltration of both vocal processes (Fig. 10). X-ray examination showed an extensive lesion of left upper lobe with several small cavitations. Sputum was positive.

Clinical course: An attempt was made to induce pneumothorax on the left side, but was unsuccessful because of adherent pleura. Patient was treated conservatively for five months, during which time she gained fifteen pounds, but the sputum continued positive. She still complained at this time of hoarseness and some dysphagia and on examination the larynx showed increased infiltration at the posterior section of the larynx.

A phrenic crush was performed September 1, 1937, and soon afterwards the patient showed definite clinical improvement. Sputum became negative November 16, 1937, and the pulmonary lesion showed fibrosis and clearing (Fig. 9). The laryngeal lesion also cleared and now appears normal (Fig. 11).

This case illustrates a subacute laryngeal lesion which cleared completely following a good result obtained through a successful operation on the phrenic nerve.

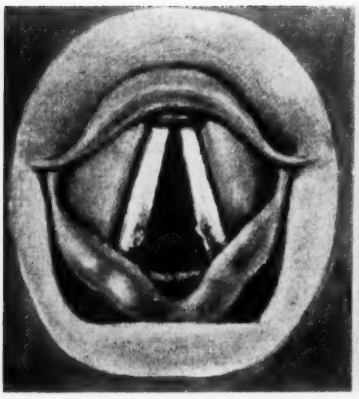

Fig. 10.

Fig. 11.

Case 3. Subacute tuberculosis of the larynx.

The chronic type, which offers the best prognosis, is characterized by firm infiltration of part or parts of the larynx, with a pronounced tendency or fibrosis and healing. The lesion, due to an excessive proliferation of connective tissue cells, is usually limited in extent and may remain so for an indefinite period. These patients suffer very little as far as the larynx is concerned and often with the exception of irritation and dryness in the throat and perhaps some hoarseness, have no symptoms at all. This type of laryngeal tuberculosis is very often overlooked. The prognosis as to life and voice is very good unless the disease becomes acute or subacute when the prognosis becomes modified.

The following case illustrates the above type:

REPORT OF CASE

Case 4.—E. J. K., male, age 46. First came under my observation April 9, 1923, at which time he had a pulmonary lesion moderately advanced with involvement of both upper lobes. Sputum was positive May 16, 1923, and after that positive and negative on and off. At the time of the first examination he was hoarse and aphonic but never had dysphagia. Examination of the larynx showed an infiltration of the left vocal cord, also of the posterior commissure. The laryngeal involvement remained more or less stationary; at times the infiltration was more severe than others. This has been going on for the past fifteen years and examination of the larynx February 16, 1938, still shows a thickening, with congestion of the left vocal cord; chronic hyperplasia of the posterior commissure. The right

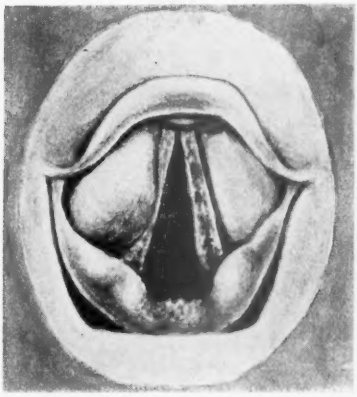

Fig. 12. Case 4. Chronic tuberculosis of the larynx.

ventricular band is hyperplastic and partly covers the right vocal cord (Fig. 12). The patient is still moderately hoarse but otherwise has had no difficulty with the larynx.

An x-ray of the chest taken February 15, 1938, shows a chronic productive lesion involving both upper lobes and especially the left, with a great deal of scar tissue formation here and there (Fig. 13).

Summary: This case definitely illustrates the chronic type of laryngeal tuberculosis, which remains more or less stationary for years and which apparently does not influence the prognosis as to life in any way.

SUMMARY

Of these above described types, the peracute type occurs in general miliary or fulminating type of hematogenous tuberculosis. The infection undoubtedly occurs by way of the blood stream. The condition is extremely serious and the prognosis is hopeless. The acute type usually occurs in the hematogenous and sometimes in broncho-pulmonary tuberculosis. The infection here is probably also through the blood stream and the outcome in these cases will depend much on the outcome of the pulmonary lesion. The subacute and chronic types of laryngeal tuberculosis are generally found in the broncho-pulmonary cases of tuberculosis, and these clinical forms of laryngeal tuberculosis probably occur through the invasion of the laryngeal mucous membrane. It may be well to stress that, although all cases of laryngeal tuberculosis can be placed in one of

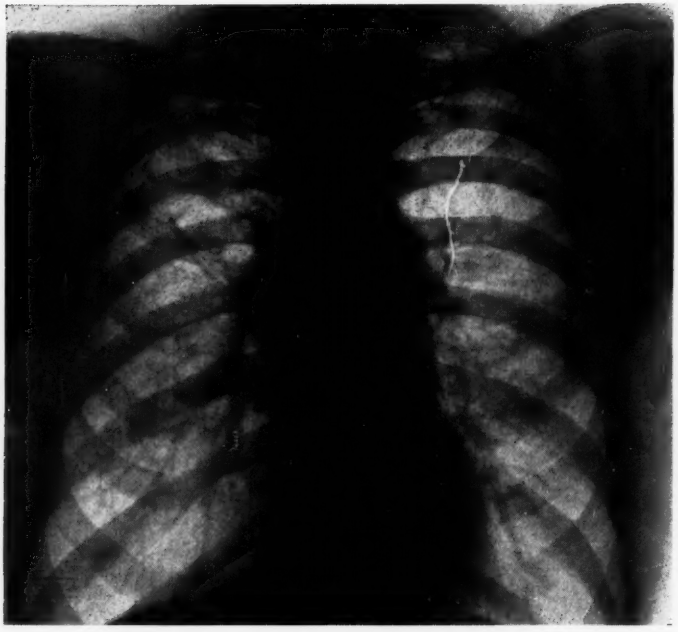

Fig. 13. Case 4. E. J. K. Pulmonary tuberculosis, chronic, productive, complicated by chronic tuberculosis of the larynx of at least 15 years' standing without much change in the character of the lesion (Fig. 12). Sputum, which was originally positive, has been negative during the past two years.

the above groups, the classification is not at all static and with the exception of the cases of the peracute type which invariably terminate fatally, all the other types may be transitional; one type may evolve into another and vice versa. An acute laryngeal lesion may become subacute and eventually chronic while the chronic case may become acute, subacute and sometimes even peracute, depending on what is happening to the pulmonary lesion.

FREQUENCY OF OCCURRENCE

In 1914[7], when I examined 500 adult patients with pulmonary tuberculosis at the Otisville Sanatorium, I found that 128, or 25.6%, of these patients had laryngeal tuberculosis. In 1934[8], twenty years later, I examined a similar group of patients at the same Sanatorium

and found that only seventy-three, or 14.6%, showed laryngeal involvement; a reduction of 11%. This, I felt, was probably due to the improved methods of treatment and especially to collapse therapy, which in 1934 was applied to 20% of the patients at the Otisville Sanatorium, also to improved diagnostic methods, such as made by the advent of fluoroscopy and x-ray, which were not available in 1914. Here it may be well to explain that the studies conducted at the Otisville Sanatorium in 1914 as well as in 1934, were made on cases of chronic pulmonary tuberculosis, the vast majority of which were broncho-pulmonary cases. There was undoubtedly a small proportion of hematogenous cases, too, but at that time these groups were not differentiated. In hematogenous tuberculosis, however, and especially in the fulminating type the story is different. During the past two years I have observed twenty cases of hematogenous tuberculosis of which sixteen (80%) showed laryngeal involvement. While this is only a small number of cases upon which to base a conclusion, it would seem that the occurrence of laryngeal tuberculosis in the hematogenous cases is very frequent and perhaps as frequent to-day as it was twenty or thirty years ago.

FREQUENCY OF OCCURRENCE OF LARYNGEAL TUBERCULOSIS

ALL CASES OF PULMONARY TUBERCULOSIS

		Larnyx Positive	
1914	500 Cases	128 Cases	25.6%
1934	500 Cases	73 Cases	14.6%

HEMATOGENOUS PULMONARY TUBERCULOSIS

1937	20 Cases	16 Cases	80%

DIAGNOSIS

Once a diagnosis of pulmonary tuberculosis has been established, the physician is on the alert, and should laryngeal tuberculosis be present such a diagnosis is easily made. However, when the patient complains primarily of the larynx, the diagnosis may be quite difficult and the laryngologist, seeing the patient for the first time, is at a disadvantage. This is especially true of the hematogenous cases, for except in the generalized miliary and in the fulminating hematogenous cases, the pulmonary symptoms are very few, and physical signs are often lacking. Even the sputum is frequently negative and only careful x-ray examination may reveal evidence of a pulmonary infection. I can recall several of such cases where physicians

had great difficulty in making a diagnosis of laryngeal tuberculosis and had to resort to biopsies. For this reason when a patient presents himself with a laryngeal lesion, such as edema, with or without ulceration of part or parts of the larynx, he should have a thorough physical examination. This should include (1) a careful history, (2) chest examination, (3) sputum analysis, (4) fluoroscopy and x-ray and (5) sedimentation test. If the patient has tuberculosis, such an examination will generally reveal it. On the other hand, if the examination is negative we can then think of carcinoma, syphillis and other conditions simulating laryngeal tuberculosis.

Although all cases of tuberculosis of the larynx are caused by the tubercle bacillus and are invariably secondary to pulmonary tuberculosis, it is well to distinguish hematogenous laryngeal tuberculosis from tuberculosis of the larynx due to a surface infection. This will help in rendering a prognosis and also aid us in deciding upon the method of treatment. The following are the distinguishing points between the two types of laryngeal infection:

Hematogenous	*Surface Infection.*
1. The onset is acute; first symptoms usually referable to the larynx.	1. The onset is insidious; first symptoms referable to the lungs.
2. On chest examination, physical signs are usually scant and often absent.	2. Physical signs usually present.
3. Sputum may be negative and especially in the beginning.	3. Sputum invariably positive.
4. X-ray examination shows characteristic hematogenous distribution.	4. Characteristic bronchogenic involvement with cavitations.
5. Marked dysphagia and often aphonia are the pronounced symptoms.	5. Dysphagia and dysphonia usually absent. Hoarseness is the commonest symptom.
6. Laryngoscopic examination —diffuse swelling of epiglottis and arytenoids, often with ulcerations.	6. Lesion usually limited to portion of the larynx and most often to the posterior commissure or vocal cords.
7. Involvement of the pharynx, soft palate, tonsils and uvula is frequent.	7. Such involvement is extremely rare.

8. Prognosis is generally poor and especially in the peracute cases where life usually extends from a few weeks to a few months, often terminating in meningitis.	8. Prognosis is usually good and especially where collapse therapy is successfully applied.
9. Treatment in the peracute cases is of no avail, and is only palliative.	9. Usually yield to treatment.

PROGNOSIS

The prognosis usually pivots around the pulmonary lesion. When the pulmonary lesion improves, no matter what type of tuberculosis it may be, the laryngeal lesion improves also. Occasionally, however, we may see a case where the pulmonary lesion seems to be improved and the laryngeal lesion is progressing. This, to my mind, is illusory and happens only because it is sometimes very difficult to ascertain the true condition of the pulmonary lesion in spite of all our improved methods of examination. Although as far as can be told the pulmonary lesion may appear improved, I believe that in such cases a certain amount of pathologic activity undoubtedly exists.

Generally speaking, the prognosis is invariably bad in the peracute cases complicating a generalized miliary tuberculosis or a fulminating hematogenous lesion. The patient is doomed and life seldom extends beyond two months, usually terminating in meningitis. In the acute cases, complicating hematogenous or exudative tuberculosis, the prognosis is better and once the pulmonary lesion is brought under control the larynx, too, will get better with and sometimes without local treatment.

In the subacute and chronic types of laryngeal tuberculosis which occur mostly in broncho-pulmonary tuberculosis and where the infection generally occurs through the surface of the mucous membrane, the prognosis is generally good. Here especially does the prognosis of the laryngeal lesion depend on the outcome of the pulmonary lesion and since the prognosis in the chronic broncho-pulmonary cases is now so much improved through the use of collapse therapy, which is now so frequently employed, the prognosis in these laryngeal cases is proportionately benefited. In other words, in the peracute and acute types where the infection is undoubtedly through the blood stream and is systemic, the prognosis is poor,

except in some of the hematogenous cases where the pulmonary lesion retrogresses, there the laryngeal lesion becomes localized and also retrogresses. In the subacute and chronic types where the infection is mostly through the mucous membrane of the larynx and remains more or less localized, the prognosis is good, and in these cases the prognosis becomes especially good when collapse therapy is successfully applied.

The voice in laryngeal tuberculosis becomes affected mostly in those cases where there is involvement of the vocal cords, posterior commissure, anterior commissure and ventricular bands. The outcome on the voice will depend mainly on the amount of structural damage and upon the mode of healing. If the lesion heals by resolution, as sometimes tuberculosis does heal, there may not be any functional disturbance whatsoever. On the other hand, if healing is by fibrosis there will be formation of scar tissue and the amount of hoarseness will depend upon the amount of structural damage.

TREATMENT

Since tuberculosis of the larynx is nearly always secondary to pulmonary tuberculosis, all our efforts should be directed towards the treatment of the pulmonary lesion. As stated previously, the healing of the laryngeal lesion invariably depends on the healing of the pulmonary lesion. It is of paramount importance to do everything possible for the control and cure of the pulmonary lesion. However, it is not within the scope of this paper to discuss the treatment of pulmonary tuberculosis, but since laryngeal tuberculosis is so intimately associated with pulmonary tuberculosis it is not possible to speak of one without discussing the other, and I will therefore mention in passing the cardinal methods universally accepted in the management of pulmonary tuberculosis.

Rest is the chief underlying principle in physiotherapy and should be employed in all forms of tuberculosis which show any evidence of clinical or even pathological activity. The kind and the amount of rest will depend mainly on the degree of activity of the lesion. From the principle of rest collapse therapy was really derived since it is a mechanical way of supplying local rest to the affected lung. Of all the remedial agencies applied in pulmonary tuberculosis, I believe that collapse therapy has done more for the prevention and cure of laryngeal tuberculosis than all the other measures combined. Of the principal forms of collapse therapy we have:

1. Artificial pneumothorax—the most common and most efficient of all.

2. Oleothorax—a method still used very sparingly in the United States.

3. Operations on the phrenic nerve—either as an independent measure or else in combination with other surgical measures.

4. Pneumonolysis—Always after a pneumothorax has failed to close a cavity.

5. Thoracoplasty, partial or complete—usually when the above surgical measures have failed or else could not be applied.

Dietetic and hygienic treatment, of course, must never be overlooked.

As to local treatment of laryngeal tuberculosis I find that the more intensely we apply treatment to the pulmonary lesion the less need is there for local therapy. In cases where collapse therapy is successful I practically never find it necessary to apply local treatment. When the cavities close and the sputum becomes negative, the laryngeal lesion invariably clears.

In those cases where collapse therapy has failed or is not feasible, it is my opinion that it is best to suit the local treatment of the larynx to the type of the laryngeal infection.

In the peracute type which usually accompanies generalized miliary tuberculosis or the fulminating type of hematogenous tuberculosis, treatment is of no avail. Here therapy is mostly palliative and is mainly concerned with relief of dysphagia. Topical application of chalmoogra oil, cocain hydrochloride sprays and also alcohol injections of the superior laryngeal nerves is about all one could do. The cautery does not aid these lesions, and as a matter of fact, in my experience renders them worse. It increases the ulceration and promotes the spread of the lesion.

The acute cases which generally occur in the less fulminating hematogenous and also in some of the broncho-pulmonary cases lend themselves more to local treatment. Here the cautery is often of great help. Vocal rest should be enforced.

The subacute and chronic cases which occur mostly in broncho-pulmonary cases I find, need very little active local treatment. This group of patients fortunately often lends itself to collapse therapy (about 50%) and it is remarkable to note the improvement in the larynx that follows a successful collapse. I have seen laryngeal lesions clear within a few weeks without leaving a trace and without ever recurring. In cases where collapse therapy cannot be applied and where the lesion in the larynx remains localized and interferes

with the functional activity of the larynx, cauterization is often of great aid. Chalmoogra oil applied locally or in a form of a spray often brings comfort to these patients. Quartz light therapy is now being employed but in my limited experience with this agency, is still of dubious value.

SUMMARY

1. Pulmonary tuberculosis is conveniently grouped in two types: Predominately broncho-pulmonary and predominately hematogenous.

2. Tuberculosis of the larynx is nearly always secondary to pulmonary tuberculosis.

3. In the broncho-pulmonary cases, about 75% of all cases of pulmonary tuberculosis, tuberculosis of the larynx occurs mainly through surface infection sometime after the development of an active pulmonary tuberculosis.

4. In the hematogenous pulmonary cases, about 25% of all pulmonary tuberculosis, the larynx becomes involved by way of the blood stream and appears either simultaneously or soon after the active dissemination occurs in the lungs.

5. A lymphogenic route probably exists but is difficult to prove.

6. Tuberculosis of the larynx can conveniently be classified into four clinical types: peracute, acute, subacute and chronic.

7. The peracute and acute types occur mostly in hematogenous pulmonary tuberculosis.

8. The subacute and chronic types occur chiefly in the broncho-pulmonary cases.

9. The incidence of laryngeal tuberculosis is much diminished (from 25.6% to 14.6%) in all cases of pulmonary tuberculosis mainly because of the application of collapse therapy, but is still very high (80%) in hematogenous tuberculosis.

10. The prognosis is invariably hopeless in the peracute cases while in all the others it depends entirely on the outcome of the pulmonary lesion. Where the pulmonary lesion improves the larynx improves also.

11. Therapy should be directed mainly at the pulmonary lesion in a most thorough manner.

12. Laryngeal tuberculosis in broncho-pulmonary cases successfully treated by collapse therapy presents a good prognosis.

13. In peracute cases the treatment of the larynx is palliative.

14. Cases receiving collapse therapy will need very little if any local treatment.

15. In all the other clinical types where collapse therapy cannot be employed, vocal rest, cautery and chalmoogra oil are our best remedial agents.

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XL

CALCIUM CEVITAMATE IN THE TREATMENT OF ACUTE RHINITIS

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The discovery and synthesis of vitamin C has made possible a significant advance in calcium therapy sufficient to warrant re-examination of the value of calcium therapy in otolaryngology.

Vitamin C possesses the unique property of solubilizing and ionizing calcium to a degree not previously attainable by oral or intramuscular administration. In calcium cevitamate, the calcium salt of vitamin C, there is available a more effective approach to calcium therapy than that of the previously used gluconate.

A comparison of the two products showed the following:

TABLE I

Salt	Per Cent Solution by Weight	Concentration Moles/Liter	Temp. Drop °C	Per Cent Ionization	Yield Ca Ion Per Gram Salt	Yield Ca Ion Per cc. Solution
Cal. Cevitamate	5%	.123	.577	74.6	.0700	.0037
Cal. Cevitamate	10%	.261	1.085	60.0	.0563	.0065
Cal. Cevitamate	20%	.589	2.362	58.1	.0545	.0137
Cal. Cevitamate	30%	1.008	3.940	55.3	.0528	.0226
Neo-Gluconate	10%	.125	.565	*47.8	.0323	.0048
Neo-Gluconate	20%	.273	1.120	*40.3	.0273	.0088
Calglucon	3%	.072	.263	48.6	.0452	.0014

*Since the structure of the neo-calgluconate is not known, and since its empirical formula is $(C_{36}H_{64}O_{38})Ca_2$, we have assumed that it ionizes as follows:

$Ca_2C_{36}H_{64}O_{38}$ ——— 2 Ca $2(C_{18}H_{32}O_{19})$

An analysis of these findings would tend to indicate the calcium cevitamate in 5 per cent solution was approximately equivalent to the neogluconate in 10 per cent solution and that the 10 per cent calcium cevitimate was approximately equivalent to the 20 per cent neogluconate.

The comparative series was run only to 30 per cent calicum cevitamate because the neogluconate solutions do not exceed 20 per cent. In considering the calglucon tablet which is only 3 per cent soluble the yield of calcium ions per cc. of solution was .0014,

whereas that of the calcium cevitamate in only 30 per cent solution was .0226, or about twenty times greater. If calculated on the basis of 100 per cent it would be in the vicinity of sixty times greater in calcium ion content per cc. solution. How important this factor can be in the question of calcium absorbability becomes apparent from the volume of solute necessary to obtain an equal amount of available calcium. For every 100 cc. of solute required for the absorption of calcium cevitamate, it would require 6,000 cc. of solute for an equal availability of calcium in calcium gluconate. Since the total fluid content of the gastro-intestinal canal is about 5,000 cc. and absorption of calcium occurs only in the upper intestine, the difference in absorbability between calcium cevitamate and calcium gluconate becomes apparent.

The action of cevitamic acid on the ionization of calicum opens up a field both of theory and practice. A comparison of the physiologic action of cevitamic acid and calcium shows an almost completely parallel action in bone metabolism, hemorrhagic diathesis, cell membrane permeability and detoxicating action. So close is this resemblance that one can interchange their functions. It is this element which suggests that cevitamic acid may be the factor which acts as the vehicle for the diffusible fraction of the serum calcium. There is a strong probability that the parathyroid hormone and cevitamic acid jointly balance the small fraction of ionized diffusible calicum. Greenwald is quoted by Cantarow as suggesting that some of the calcium is bound to an organic substance in a citrate-like combination, the compound being intimately connected with the parathyroid hormone, probably diffusible and slightly ionized. Belief in the existence of such a compound is also shared by Klinke and by Sendray and Hastings. The latter drew attention to the similarity of action of the parathyroid hormone and citrate solutions in holding calcium in solution. In view of the fact that calcium citrate is only 0.8 per cent soluble, it is difficult to conceive of citrate as a calcium solubilizing agent. Cevitamic acid, however, has precisely the desired action on the ionization of calcium and its distribution in the intestinal canal, pituitary and adrenal makes it a much better hypothetical agent than citrate.

From the practical aspect, it is important to note that the injection of calcium cevitamate is nonirritating and better tolerated than gluconate.

In considering the action of calcium cevitimate in the series of cases here reported, one must take cognizance of the vitamin C action and its time-honored use as citric drinks in the treatment of the com-

mon cold. However, if my theory as to the function of cevitamic acid is correct, one must attribute the ultimate benefit to calcium action.

A series of clinical trials were undertaken. All acute upper respiratory tract infections that applied for treatment in the ambulatory state and were free from fever, were considered in the group of the common cold.

Some were associated with acute exacerbations of chronic previously treated sinusitis, frequently of influenzal character; others were of the simple infectious type associated with obvious exposure to persons suffering from cold.

The character of the response to treatment with calcium cevitamate is strikingly shown in the accompanying chart. The cases relieved by one injection were considered well in 24 hours or given a second injection on the second or third day. Cases associated with sinusitis received a continuation of the injections for one or two weeks because of the marked beneficial effects of the calcium cevitamate. Patients were advised to appear for treatment as close to the onset of the cold as possible, and were instructed to be mindful of the beginning dryness and discomfort of the nasopharynx and a fresh post-nasal discharge. These symptoms were observed to precede the coryza by about 12 to 24 hours. In a series of over two thousand injections, there were no complications incident to the injection. With but few exceptions there was no pain or reaction. There were, however, some instances of pain such as are incident to the injection of 3 cc. of any fluid, and soreness on pressure for a few days. This latter condition varied with the location of the injection, being absent if placed deep, slightly above the upper outer quadrant of the gluteus. Although a much larger series of injections was given than are here recorded, only cases of upper respiratory conditions affecting the nose and ear are presented. The period of study covered two years and included a hundred cases. Some of the patients returned promptly for injections at the onset of or during a cold, several times during the year. In the acute cold, the injections were given daily in alternating sides, or every other day for one to four injections. In cases associated with sinusitis, two injections were given for the period of the treatment. Allergic patients were given two injections weekly, supplemented by 10 to 15 grains of nucleic acid three times daily. Improvement was noted as moderate, marked or complete. It would seem an extravagant statement to say that all cases showed improvement, yet with but a few isolated exceptions, all benefited by the reduction in nasal congestion and discharge and

rapidity of recovery. The usual period of exhaustion following a cold was remarkably shortened.

The accompanying list of case reports represents 100 cases in continuous series treated with calcium cevitamate. In no case was there any complication associated with the injection. There were no sloughs, and only one case showed, on repeated injection, a tendency to lumpiness which gradually disappeared, thus establishing the non-irritating character of the solution and its ready absorption. Although in this series of cases 296 injections were administered intramuscularly, over 2,000 injections of the calcium cevitamate have been administered without any complications.

The cases reported comprise those of upper respiratory tract infection, of which 48 were the common cold, 38 were combined acute rhinitis and sinusitis and two acute laryngitis, 12 cases had nasal allergy. The tabulation of results showed 42 per cent completely relieved, as a rule after the first or second injection, comprising chiefly the cases of acute cold, 35 per cent of which were markedly improved comprised chiefly the cases of acute cold combined with sinusitis, and 3 per cent but moderately improved comprised the more or less chronic types. The allergic cases responded remarkably well.

In presenting this work on clinical material, one is mindful of the fact that animal experimentation would be valuable. However, since the etiologic agent of the common cold has not yet been isolated, there remains no other control than clinical application which in this study gave conclusive and gratifying results.

SUMMARY

1. The value of calcium therapy in otolaryngology has been enhanced through the use of the calcium salt of vitamin C, the calcium cevitamate.

2. A comparative test of the physical properties of calcium cevitamate and calcium gluconate shows a marked difference in solubility and ionization.

3. Since an ampule of 3 cc. of a 15 per cent solution of calcium cevitamate was injected, each dose represented about 450 mgs. of vitamin C. The calcium content is about 11 per cent when calculated as calcium oxide.

4. Calcium cevitamate would appear to be practically an abortive in the treatment of the common cold.

351 WEST 86TH STREET.

TABLE II

Case #	Patient	Sex	Date	Diagnosis	Comment
1	B. B.	M	3 24, 1937	Acute cold	Marked improvement
2	C. G.	F	4 5, 1937	Acute cold	Complete relief
3	E. K.	F	4 5, 1937	Acute cold	Complete relief
4	J. G.	F	4 5, 1937	Acute cold	Complete relief
5	I. G.	F	4 5, 1937	Acute cold Rt. ethmoiditis	Marked improvement
6	H. S.	F	4 1, 2, 5, 7, 1937	Grippe, left Sphenoiditis	Marked improvement
7	P. D.	M	4 6, 1937	Aphthous stomatitis. Acute cold	Complete relief Stomatitis disappeared afteer one injection
8	G. B.	M	4 7, 1937	Acute laryngitis	Marked improvement
9	L. G.	F	4 7, 12, 1937	Acute cold	Complete relief
10	D. S.	F		Bilateral maxillary and ethmoidal sinusitis	Improvem't noted with each injection
11	L. K.	M	11 9, 11, 1936	Acute rhinitis Bilateral ethmoiditis	Marked improvement
12	P. S.	F	11 10, 15, 12 7, 1936 3 31, 1937	Bilateral ethmoiditis	Marked improvement
13	W. H.	M	4 20, 1937	Acute cold	Complete relief
14	R. R.	F	4 27, 1937	Bilateral ethmoiditis	Moderate improvement
15	F. G.	F	4 27, 1937	Bilateral ethmoiditis	Marked improvement
16	D. D. D.	M	4 28, 1937	Acute rhinitis	Marked improvement
17	M. B.	F	5 3, 7, 1937	Bilateral ethmoiditis	Marked improvement
18	L. L. M.	M	5 10, 1937	Acute rhinitis	Complete relief
19	G. B.	F	3 1, 16, 4 6, 1937	Acute cold	Complete relief
20	H. C.	F	3 20, 21, 1937	Bilateral maxillary sinusitis	Marked improvement
21	R. M.	F	3 21, 1937	Acute cold	Complete relief

TABLE II—(Continued)

Case #	Patient	Sex	Date	Diagnosis	Comment
22	J. O'D.	M	3 20, 1937	Acute cold	Complete relief
23	J. N.	M	3 23, 24, 31, 1937	Bilateral maxillary sinusitis	Marked improvement
24	E. S.	F	3 25, 1937	Acute cold	Complete relief
25	R. G.	F	3 28, 1937	Acute cold	Complete relief
26	A. S.	M	10 21, 25, 11 13, 12 21, 1936; 1 15, 29, 2 24, 25, 1937	Acute rhinitis; deflected septum, ethmoid, polypoid	Marked improvement
27	C. R.	F	10 22, 11 12, 1936	Acute rhinitis	Completely relieved
28	H. R.	M	10 23, 26, 11 27, 30, 1936; 1 8, 18, 22, 29, 2 24, 1937	Acute rhinitis Bilateral maxillary ethmoidal sinusitis	Acute symptoms relieved; sinusitis improved
29	N. K.	F	10 30, 11 2, 4, 6, 11, 12 19, 1936	Vasomotor rhinitis, severe	Marked improvement
30	E. J.	F	11 2, 9, 13, 17, 1936	Bilateral ethmoiditis	Marked improvement
31	S. W.	F	11 2, 1936	Acute cold	Complete relief
32	W. J. R.	M	11 3, 9, 23, 1936	Acute rhinitis Bilateral ethmoiditis	Marked improvement
33	A. S.	F	11 3, 9, 11, 14, 1936	Deflected septum Bilateral ethmoiditis	Marked improvement
34	S. T.	F	10 29, 11 3, 10, 25, 1936	Bilateral ethmoiditis; spheno-palatine ganglion neuralgia	Marked improvement
35	G. F.	M	11 4, 1936	Acute cold	Complete relief
36	A. G.	M	11 4, 1936	Acute rhinitis	Complete relief
37	S. R.	M	10 16, 1936	Acute rhinitis	Relieved completely
38	S. H.	M	10 15, 23, 26, 1936; 1 8, 3 10, 12, 1937	Acute rhinitis Bilateral ethmoiditis	Relieved completely

TABLE II—(Continued)

Case #	Patient	Sex	Date	Diagnosis	Comment
39	E. L.	M	10/20, 1936	Acute rhinitis Left ethmoiditis	Marked improvement
40	T. C.	F	10/20, 23, 27, 11/9, 17, 12/9, 14, 21, 1936; 1/5, 8, 1937	Bilateral ethmoiditis	Marked improvement
41	L. A.	F	10/20, 23, 26, 11/7, 12/11, 1936; 2/27, 1937	Acute rhinitis, deviated septum, bilateral ethmoiditis	Relieved of acute attacks. Sinusitis improved
42	F. R.	F	10 10, 21, 1936	Acute rhinitis	Relieved completely
43	A. C.	M	10/21, 24, 26, 11 6, 9, 11, 12, 1936	Acute rhinitis Bilateral ethmoiditis	Marked improvement
44	F. S.	M	10/21, 25, 28, 12/14, 1936; 1/27, 3/10, 12, 4/7, 1937	Acute rhinitis, left pan-sinusitis; left radical pan-sinus operation 4 yrs. ago	Marked improvement
45	I. E.	M	10 21, 1936	Acute rhinitis	Relieved completely
46	E. E.	F	10/21, 28, 11/4, 1936	Acute rhinitis Bilateral ethmoiditis	Completely relieved
47	L. C.	F	10 22, 26, 29, 11 23, 27, 1936	Acute rhinitis Bilateral ethmoiditis	Relieved completely
48	J. W.	M	11 9, 12, 1936	Acute rhinitis	Marked improvement
49	A. F.	F	10/23, 26, 11 17, 23, 1936	Acute rhinitis; nasal allergy	Marked improvement
50	A. K.	F	10/26, 1936	Acute rhinitis Bilateral ethmoiditis; left sphenoiditis	Marked relief
51	B. S.	F	10/26, 1936	Acute rhinitis	Completely relieved
52	L. S.	F	10 26, 1936	Acute rhinitis; right maxillary sinusitis, old radical operation	Marked improvement

TABLE II—(Continued)

Case #	Patient	Sex	Date	Diagnosis	Comment
53	C. S.	M	10/26, 28, 1936; 1/5, 8, 1937	Acute cold; biilateral ethmoiditis	Marked improvement
54	S. C.	F	10/26, 11/9, 1936	Acute rhinitis	Marked improvement
55	R. S.	F	10/27, 30, 1936	Acute cold; hypertrophic turbinates	Marked improvement
56	M. R.	F	10/27, 1936	Acute cold	Marked improvement
57	B. B.	F	10/18, 1936	Acute cold	Complete relief
58	Mr. G.	M	10/28, 1936	Bilateral ethmoiditis	Marked improvement
59	B. N.	M	10/28, 11/2, 9, 23, 12/7, 21, 1936; 1/4, 15, 1937	Acute rhinitis	Complete relief
60	F. W.	M	4/1, 1937	Acute cold	Complete relief
61	L. L.	M	4/9, 1937	Vasomotor rhinitis	Marked improvement
62	H. W.	M	4/9, 12, 19, 1937	Acute cold	Marked improvement
63	A. C.	F	4/10, 1937	Bilateral ethmoiditis, acute rhinitis	Marked improvement
64	M. R.	F	4/10, 13, 1937	Acute cold	Complete relief
65	R. M.	M	4/3, 1937	Acute cold	Improved after one injection
66	R. L.	F	4/5, 15, 1937	Bilateral ethmoiditis	Marked improvement
67	D. L.	M	4/13, 1937	Acute rhinitis	Marked improvement
68	K. M.	M	4/13, 1937	Acute rhinitis	Complete relief
69	N. F.	F	4/11, 13, 1937	Acute cold	Complete relief
70	S. L. R.	M	4/19, 1937	Acute cold	Complete relief
71	S. F.	M	4/1, 19, 1937	Acute cold	Complete relief
72	M. H.	M	4/19, 23, 27, 30, 1937	Billat. polypoid, asthma ethmoid. maxillary sinusitis	Marked improvement

TABLE II—(Continued)

Case #	Patient	Sex	Date	Diagnosis	Comment
73	G. N.	M	4/24, 1937	Bilateral ethmoiditis	Complete relief
74	E. H.	F	4/26, 29, 1937	Acute cold	Complete relief
75	J. F.	M	8/18, 1937	Allergic rhinitis	Marked improvement
76	A. B.	F	8/18, 1937	Acute otitis, grippe type Acute rhinitis	Improved. No paracentesis
77	M. R.	F	6/25, 28, 1937	Acute rhinitis	Complete relief
78	J. B.	M	10/28, 1937	Acute cold	Complete relief
79	A. S.	M	7/8, 13, 8/16, 1937	Nasal allerty; Bilat. maxillary and ethmoidal sinusitis	Marked improvement
80	B. L.	F	7/12, 16, 1937	Acute rhinitis and bronchitis	Marked improvement
81	I. L.	F	7/17, 8/9, 25, 9/16, 20, 24, 27, 10/1, 3, 7, 15, 18, 19, 1937	Bilateral ethmoiditis	Moderately improved
82	L. S.	F	7/17, 23, 8/4, 6, 13, 20, 9/8, 12, 28, 1937	Bilateral ethmoiditis	Marked improvement
83	L. I.	M	4/9, 1937	Allergic rhinitis	Marked improvement
84	A. C.	F	4/10, 1937	Bilateral ethmoiditis	Marked improvement
85	S. F.	M	4/1, 19, 1937	Acute cold, postnasal drip	Marked improvement
86	R. L.	F	4/5, 17, 1937	Bilateral ethmoiditis. Sinusitis	Moderately improved
87	F. W.	F	11/7, 1936	Acute cold	Complete relief
88	L. S.	M	11/7, 9, 1936	Acute cold	Complete relief
89	M. R.	M	11/7, 1936	Acute cold	Complete relief
90	O. F.	M	10/28, 1036	Acute rhinitis	Complete relief
91	I. B.	F	10/28, 11/6, 16, 1936	Deviated septum, Bilateral ethmoiditis; acute rhinitis	Marked improvement

TABLE II—(Continued)

Case #	Patient	Sex	Date	Diagnosis	Comment
92	R. T.	M	10/28, 11/2, 16, 1936	Sinusitis; bilateral maxillary sinusitis. Asthma	Marked improvement in asthma and sinusitis
93	C. N.	M	10/30, 1936	Acute rhinitis	Completely relieved
94	C. A.	M	10/30, 1936	Acute rhinitis	Completely relieved
95	A. M.	M	10/31, 11/7, 21, 1936	Allergic vasomotor rhinitis	Marked improvement
96	R. M.	F	11/1, 9, 19, 12/2, 20, 1936; 1/5, 10, 15, 2/19, 25, 3/16, 1937	Post-nasal drip Bilateral ethmoiditis	Marked improvement
97	D. K.	F	10/14, 20, 11/2, 9, 1936	Allergic rhinitis. Polypoid pan-sinusitis. Deflected septum	Allergy improved greatly. Sinusitis recurred. Radical operation
98	N. G.	F	7/16, 9/27, 10/25, 1937	Bilateral maxillary sinusitis. Severe asthma, nasal allergy	Asthma completely relieved. Sinusitis markedly improved
99	J. S.	F	6/28, 7/14, 10/20, 23, 1937	Bilateral maxillary ethmoid sinusitis. severe asthma, nasal allergy	Asthma markedly improved. Sinusitis markedly improved
100	J. G.	M	8/1, 21, 24, 31, 9/1, 3, 16, 25, 10/2, 9, 16, 25, 30, 1937	Bilateral ethmoiditis. Nasal allergy. Asthma	Asthma completely relieved. Nasal allergy relieved. Sinusitis markedly improved.

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XLI

MALIGNANCIES OF THE NOSE AND THROAT*

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SAN FRANCISCO

The purpose of this paper is to set forth our experiences of the past fourteen years in the treatment of malignancies of the nose and throat. Only nineteen cases comprise the series, because no case was included unless the patient could be followed in every detail from examination and biopsy to treatment and its outcome. This is much too small a number from which to draw conclusions, but the analysis has helped us clarify our ideas of therapy.

The factors to be determined when a patient presents himself are: (1) The type of tumor; (2) the presence or absence of metastases; (3) the site of the tumor, considering the possibility of its removal surgically; and (4) the type of treatment to be chosen, bearing in mind the patient's economic status and psychological makeup, and the tumor's possible response to the use of roentgen ray or radium. To determine the latter, we review briefly the action of radiation.

The action of radium, screened so that the gamma rays alone are effective, is similar to that of the roentgen rays. The more immature the cell the greater is the effect of the rays. Therefore, cells undergoing mitotic division and those rich in nuclear chromatin are most responsive to radiotherapy. If the tumor responds there is either autolytic degeneration of the cells, which is the desired result, or an arrest of the tumor cells, so that surgical removal may have a better chance of success. The tumor bed undergoes inflammatory reaction, ending in fibrosis and an obliterative endarteritis, which reduces the supply of blood to the tumor.

Infection is held to be a contra-indication to the use of radiotherapy as the resistance of the tissue is impaired and severe sloughing may result.

The immediate effect (in three weeks) of radiotherapy is an epithelitis with erythema and small patches of fibrin on the mucosa, as well as some swelling. If, therefore, the airway is obstructed, a

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*Read before the Western Section of American Laryngological, Rhinological and Otological Society at Santa Barbara, California, January 29th, 1938.

tracheotomy is indicated before therapy is begun; otherwise what should be an orderly, leisurely procedure may be converted into an emergency tracheotomy.

Harrison and Sarasin (J. of Laryngology, 50:258 [May], 1935) advocated the use of ultra short wave therapy to decrease the symtoms of the reaction. "From the time of the commencement of the radio-epithelitis ultra short wave therapy is given, starting with seven minutes, increasing to twenty, in some cases longer. The electrodes, each about 20 by 30 cm., are placed on each side of the neck and the intensity is regulated according to the subjective feelings of the patient, being as warm as he can tolerate. These are given before the x-ray treatments. Objectively the infection is reduced and necrotic materials and membranes are shed from the tumors. At the same time edema of the soft tissues is reduced." In addition, these authors noted that, with regard to the epithelial reaction, the less intense the reaction, the longer it may be permitted to exist.

The general feeling of the radiotherapists with whom I have worked is that the tumor must be destroyed in the first series of radiations. If insufficient therapy is given initially, the tumor fails to respond well to later therapy; if the tumor is of the radio-resistant type it becomes more so by virtue of the initial series.

Cartilage may break down months after treatment, as its resistance to infection seems lowered by intensive, though protracted and fractionated, radiation. This should be borne in mind in laryngeal therapy.

All patients receiving heavy doses about the pharnyx complain of an annoying sensation of dryness which usually lasts for about a year.

So much for generalization about radiotherapy. We wish now to review our limited experiences with various types of therapy.

MALIGNANCIES OF THE LARYNX

The nomenclature is hopelessly confusing to anyone. One pathologist calls a growth "cornifying epithelioma," while another refers to the same growth as an "epidermoid" type.

We have had nine cases of laryngeal malignancy classified as follows: epidermoid carcinoma, six; premalignant multiple papillomata, one; cornifying epithelioma, two. These last two were really epidermoid in type, but were further differentiated, possessing areas of cornification.

Of the six patients in the first group, five were free from metastases, but only two were treated surgically. Of the two surgically treated, one patient, a woman 55 years old, had a small papilloma which was widely removed endolaryngeally, and has not recurred in three years; the microscopic diagnosis was epidermoid carcinoma. The second case, occurring in a male 50 years of age, showed involvement of the commissure and one cord was fixed. A one-stage laryngectomy was done and the patient is free from recurrence after nine years; the microscopic diagnosis was epidermoid carcinoma. His speaking voice is poor, but the use of an instrumental aid permits him to operate his drug store.

Two patients in whom the growth was operable were treated by roentgen therapy, using the Coutard technique. Both made their living as salesmen and were of the extrovert type. Their families were opposed to laryngectomy, feeling sure that the patients would commit suicide even though the operations were successful. The growths were situated near the anterior commissure and were not suitable for laryngofissure. Details of their therapy follow:

The first of these two patients, a man, 59 years of age, received a total of 7200 r. in sixteen treatments between November 12 and December 4, 1936. The 200 kv. radiations were used through ports 5 by 7 cm., and the filter was 2 mm. cu.

The second patient, a man, 55 years of age, had twenty-two treatments, totaling 6600 r. from April 5 to May 4, 1935; the 200 kv. apparature was used with a filter of 2 mm. cu. This patient is well and free from recurrence to date. The reaction of skin and mucosa was severe. The diagnosis was papillary carcinoma of the left cord.

A third patient without metastases was not operated on, as he was deemed a poor risk because of a severe myocarditis; the lesion itself was operable. Radon (three gold tubes of 2.2 mc. each) was implanted by direct laryngoscopy into the base of the growth on the left cord. The patient died three months later from heart disease. The cord was clear and there were no metastases to the glands. The diagnosis was epidermoid carcinoma of the larynx.

These three patients were treated according to the outline given above, not because the tumors were held to be radio-sensitive, but for other reasons.

In the sixth case the growth (epidermoid in type) was inoperable when the patient was first seen, as it involved the left pyriform

sinus, ary-epiglottic fold and the deep cervical glands. A preliminary tracheotomy was done on account of an obstructed airway. The patient was given 2,000 r. right and left, and 1,800 posteriorly over a period of 23 days; each field was 8 by 10 cm. and a unit dose of 200 r. (measured in air) was administered; the 900 kv. machine was used. Immediate recession took place, but death occurred in four months from recurrence.

The seventh case, one of multiple papillomata, occurred in a female 26 years of age. These tumors were removed four times by direct and indirect laryngoscopy from October, 1933, to May, 1935. They recurred more rapidly after each removal, and the last two microscopic examinations showed a definite premalignant type of growth, according to two excellent pathologists. Because of her youth and fine appearance, it was decided to try fractional x-ray. From June 17 to July 3, 1935, she received 4,800 r. over three areas, the 200 kv. apparatus being used with 2 mm. copper as a filter. The growths melted away, but in September there was a small papilloma near the anterior commissure on the left cord. From September 28 to October 29, 1935, she received 6,000 r. over three areas. There was no further difficulty for one year, when she developed a severe upper respiratory infection and edema of the larynx, necessitating an emergency tracheotomy by another physician; a tracheo-esophageal fistula developed, requiring the use of a permanent feeding tube for six weeks. Both tubes were finally dispensed with after purulent material, probably from a cartilagenous slough, had been coughed up through the mouth. This patient has remained well to date except for a deformed left cord and rough voice. There are multiple telangiectases of the skin over the larynx. This late breaking down of cartilage has been reported before, but this case represents our sole experience with this difficulty.

The two patients with cornifying epithelioma, both free from metastases, were subjected to laryngectomy of the one-stage type. One of these patients, who had a growth on the anterior commissure with beginning fixation of the cords, died one week after operation from a probable mediastinitis. This man, 59 years of age, was greatly overweight and was a poor operative risk. The second patient was seen in June, 1927, and operated on in August. He was a slender male, 50 years of age, who had been treated elsewhere for a year for chronic laryngitis. No biopsy had been taken, though a growth on the anterior commissure had fixed the left cord by the time he came into our hands. He is free from recurrence to date, has a good voice, and is able to work at his profession.

No generalizations can be drawn from this small series, and no claims made that roentgen treatment is to be preferred. I feel that the patient's age, economic status and mental makeup, as well as the operability of the growth, are all factors that must be considered. If the tumor is operable, surgery seems the method of choice. We have found no cases suitable for laryngofissure. In this series the most satisfactory results of roentgen therapy have been in carcinoma of the larynx.

MALIGNANCIES OF THE PHARYNX

We have had five patients with malignant tumor of the pharynx; four of them had metastases.

The patient without metastases was a physician, 64 years of age, in whom the growth occurred on the soft palate. He was first treated in February, 1925, for epidermoid carcinoma. Radon, 4.6 mc., in five glass tubes, was used. There was no recurrence until March, 1937, when a suspicious nodule appeared on the right posterior pillar at its junction with the pharynx. Two gold tubes of 1.8 mc. each were implanted and the nodule regressed. The patient is living after twelve years.

Of the patients with metastases, two had lympho-epithelioma, which is classed as a radio-sensitive tumor. In each instance the growth arose in the lateral portion of the lingual tonsil and in each there was a negative biopsy which was repeated because of the clinical evidences of malignancy. The second biopsy revealed the true nature of the growths. In both cases there were metastases to the glands and operation was impossible.

The first patient in this group, 72 years of age, had 8,250 r. from February 25 to April 25, 1936. The 200 kv. apparatus was used at a distance of 50 cm., with 2 mm. cu. and 1 mm. al. as a filter. Four ports were used. On November 6, 1936, there was local recurrence and four gold tubes of radon (1.4 mc.) were inserted. This treatment was followed by regression. On February 16, 1937, three gold tubes of 2.9 mc. were inserted, but no result was evident locally and the patient died on June 30, 1937. The reaction to the roentgen therapy was severe and the patient suffered great pain throughout the course of the disease. Death occurred sixteen months after the onset.

The second patient, 60 years of age, received 9,000 r. delivered through three ports, the 900 kv. machine being used. A submaxillary gland metastasis was present at onset of treatment and was present, though small, eight months later. The patient was first seen in

November, 1936, and is still alive, though local recurrence is present. Pain has been severe. This is another instance in which a tumor, believed to be radio-sensitive, has failed to respond to roentgenotherapy. The tumor itself did disappear for six months, though the metastasis to the gland did not.

A third case was of carcinoma of the right tonsil, with metastases to the submaxillary glands. The patient was first seen in April, 1934, when a total of 3,500 r. was given to the right side of the neck in sixteen doses. From December 5, 1935, to March 10, 1936, 2,200 r. in fifteen treatments was administered to each side of the neck. From April 24, 1936, to June 26, 1936, ten treatments, totaling 3,000 r. were given over the metastatic glands on the right side of the neck, and from August 25, 1936, to October 8, 1936, nine treatments, totaling 1,700 r., were given to each side of the neck. Then from October 22 to November 9, 1936, 2,400 r., in six treatments, was used over the metastatic glands in the submental region.

The factors in all treatments were: 200 kv. radiations, at a distance of 50 cm., Thoreau's filter, having one-half value layer of 1.65 cu., over fields 8 by 10 cm. in size, except for the last series, in which a field 5 by 7 cm. was used.

The total was 12,800 r. Death occurred in April, 1937, from progressive weakness after three years' treatment.

In the fourth case the tumor was epidermoid, probably originating in the larynx but involving the epiglottis and base of the tongue. The patient was given 5 gold tubes of 7.5 mc. on October 21, 1931. From November 11 to 16 he was given a total of 1,000 r. to each side of the neck, using a field 8 by 10 cm. From April 13 to 28, six treatments were given, totaling 1,500 r. to each side of the neck, using a field 8 by 10 cm. The factors used were 200 kv. with ½-mm. cu. and 1 mm. al. filter, at a distance of 50 cm. The total dose amounted to 5,000 r. The growth shrank to one-half its former size and then ran wild, death occurring on August 28, 1932.

SUMMARY OF CASES OF MALIGNANCIES OF THE PHARYNX

One patient treated with radium is alive after twelve years. The growth occurred on the palate and recurred in the pharynx. Three patients, all with metastases to the glands, succumbed after sixteen months, three years and ten months, respectively. The fourth patient, who has cervical metastases, is alive one year after beginning treatment, but has a recurrence and is failing rapidly.

NASAL MALIGNANCIES

We have had five cases of malignancy of the nose, for which we have complete records.

In case one the growth was called "epithelioma of the nasal mucous membrane." This patient was a man 38 years of age, seen first in April, 1928. The growth occurred in the anterior ethmoids. A thorough internal ethmoidectomy was done, and one year later biopsy was negative. This patient is still alive, though we have not seen him for six years and cannot report on the local condition.

The second patient, 62 years of age, was seen on June 5, 1935. At that time he had a small mass in the nasopharynx which was diagnosed as carcinoma of the lympho-epithelioma type of Ewing. It was curetted out and 3.2 mc. of radon was inserted. Roentgenotherapy, 11,000 r. in 32 treatments, was given between May 6, 1935, and April 29, 1937. The patient refused to believe that his condition was serious and was so irregular in taking the treatments that no single intensive series could be given. Irradiations from the 200 kv. apparatus were used, with Thoreau's filter .44 mm. tin, 25 mm. cu. and 1 mm. al., at a distance of 50 cm. over fields 8 by 10 cm. in size. No reaction of the skin occurred.

In July, 1937, this patient suffered severe pain and loss of hearing in the left ear, as well as diplopia. Roentgen films showed no changes in the base of the skull, but a total of 6,800 r. was given through three ports, the anterior left malar region, the left supra-auricular region, and the left posterior auricular region. The 200 kv. apparatus was used with a normal Thoreau's filter composed of tin, copper and aluminum with one-half value layer of 1.6 mm of copper. A total of 17,800 r. was given. The patient is still alive, but we believe that the growth will progress.

Our third patient was a female, 60 years of age. A large polyp in the left posterior ethmoids was diagnosed a lympho-epithelioma on September 11, 1935. Radium was inserted. On January 19, 1937, a specimen was taken from the posterior ethmoids and showed an "attenuated carcinoma." Radium, 23.7 mg. with ½-mm. silver and 3 mm. in rubber screen, was packed in the area for two and one-half hours. There is no evidence of recurrence at present.

The last two patients were diagnosed as having angio-endothelioma of the nasopharynx and are included in this group, since the exact point of origin could not be determined. This tumor is a type of nasofibroma, free bleeding, not truly malignant, in that it does not

metastasize and does not recur if fully removed. Because of the bleeding, surgery was not done and x-ray was used.

Case one was that of a 10-year-old boy seen on May 2, 1933. He had had a nasal fracture one year previously, followed by nasal obstruction in six months. The entire left nostril was plugged by a growth which bled copiously when touched and which had elevated the left nasal bone, giving the boy a "frog-faced" appearance. The tissue, however, came from the mass in the nose, of which the point of origin was and is unknown.

From July, 1933, to April 14, 1934, he was given 42 treatments, totaling 10,400 r. The growth showed immediate response, and by December, 1933, it could not be seen intranasally. The left nasal bone sank back into position and the face appeared normal.

This patient was last seen in September, 1937. At that time he appeared to be normal to inspection and showed no recurrence. There was crusting, as the turbinates had been destroyed by pressure, the naso-antral wall had disappeared, the front face of the sphenoid was gone and the septum lay against the right lateral nasal wall. Eventually we plan to correct the septal deformity to lessen the amount of crusting. This is one of the most dramatic cases we have seen.

The second case was in a boy, aged 10, seen in October, 1935. He had had repeated severe epistaxis, and one attempt had been made to remove "adenoids." A large tumor presented in the nasopharynx and left naris.

Roentgenotherapy totaling 10,000 r. was used, crossfiring the posterior nasopharyngeal area through three ports. The treatments were given almost daily from October 30 to December, 1935. The 200 kv. radiations were used at a distance of 50 cm., with 2 mm. cu. as a filter. In December, 1937, the boy's father reported no further nasal obstruction nor epistaxis. As the boy lives in the South Seas he was not brought in for examination.

The question seems settled in the minds of nearly all writers that the anaplastic type of tumor is the most susceptible to roentgenotherapy. It is also stated that lympho-epithelioma or the transitional cell type is amenable to such therapy. This was not borne out by our experience, as two patients with tumors of this type were treated by the Coutard technique and, in each, the growth progressed after a temporary arrest. One patient died and the other is rapidly approaching death.

Broders' classification is used widely and is the basis from which the foregoing statement was derived. One pathologist says it is of great help, another the opposite. Meanwhile the physician in charge wonders and worries as to the correct procedure.

Because of such confusion, we believe surgery is always to be preferred where it is possible anatomically, when the patient's economic and psychic status do not contra-indicate its use.

The x-ray therapy should be administered only by those of wide training and experience and the nose and throat specialist should watch the development of mucosal epithelitis, which is the best indicator of proper dosage. The reaction of the skin is secondary in importance.

Our impression is that if the initial x-ray series does not cure the new growth further therapy is merely palliative.

It must be pointed out that many other patients were seen in this interval, but were not included in this series because of incomplete records. Many patients with advanced malignancies were not subjected to biopsy for economic reasons. The inclusion of this group would serve to lower the number of arrested cases, as most of them were given x-ray or radium, but the absence of a biopsy rather that a desire to show favorable results prevented their being reported.

SUMMARY

We have had nine cases of malignancy of the larynx. Four of these patients were operated on; three are living and well after three years, nine years, and ten years, respectively. One patient died a week after operation, probably from mediastinitis. Four patients were treated by the Coutard technique of roentgenotherapy. Three of these are living, without recurrence, at present, having survived one year, two years and six months, and two years and eight months, respectively. One died four months after treatment. Radon was used for only one patient, in whom death occurred from other causes than the growth.

Five patients with malignancy of the pharynx are included in this series. None was operated on. Four were treated by x-ray (Coutard technique), three of whom died in ten months, one year and four months, and four years, respectively. One patient is living one year after treatment, but recurrence is present. One patient, without metastases, was treated by the use of radon and is living and well after twelve years.

Of the five patients with malignancy of the nose, one was operated on and has remained well for six years. One received a combination of radon and x-ray therapy and is living after two years and six months, but recurrence is probable in this case. Two were treated by roentgenotherapy, both of whom are living and well after two years and three months, and four years and seven months. One was treated by radium and is alive and well after two years and five months.

Of the nineteen patients in the whole series, eight are living and free from recurrence; two are living with recurrence; eight died from malignancy; and one died from other causes.

384 Post Street.

XLII

VARIX OF THE VOCAL CORD

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DETROIT

In the review of the clinical and pathological reports of Harper Hospital, Detroit, no mention can be found of a condition affecting the vocal cord and giving the clinical and pathological characteristics of a true varix.

Hemorrhage into the vocal cord is not unusual. However, a true dilatation of a blood vessel with thrombosis is rare. It would seem that the cause of each condition is loud speech, necessary above the noise of moving machinery, particularly by those who have to use the telephone under such circumstances. The sudden sharp blast of air traumatizes the cord sufficiently to produce the lesion which in one person is a hemorrhage and in another, dilatation with thrombosis. This latter is a true varix of the vocal cord.

Some people are predisposed to such dilatations of the cord vessels. Age, chronic irritation, local and general obstructions, all play their part in causing the vessels to dilate. When hemorrhage into the tissues of the cord occurs, swelling of the cord results. If this small mass be removed at the proper stage, the remains of the hemorrhagic extravasation in the form of phagocytical hematoidin crystals and hyperkeratosis is seen. Disappearance of this blood may be followed by some contraction of the cord tissues but without serious clinical results. According to Chevalier Jackson, even a slight concavity of the cord may well make a difference in vocalizing, in those whose voice is used for finer tones. But, in the factory worker, where the condition is most frequently seen, this change is of no functional importance.

REPORT OF A CASE

CASE 1. The patient was a man of 50, an automobile factory worker. His physical findings were negative except for a hoarseness of one year's duration, previous to examination, on December 12, 1936. This examination revealed the lesion of the larynx, which is the subject of this report. It appeared as a purplish red, sausage-shaped tumor projecting sufficiently into the rima to produce hoarseness. So far as could be seen, it arose from the margin of the cord. It gave the impression that this was a condition differing from what was usually seen in this region. On January 6, 1937, the patient was operated upon. The operative procedure was

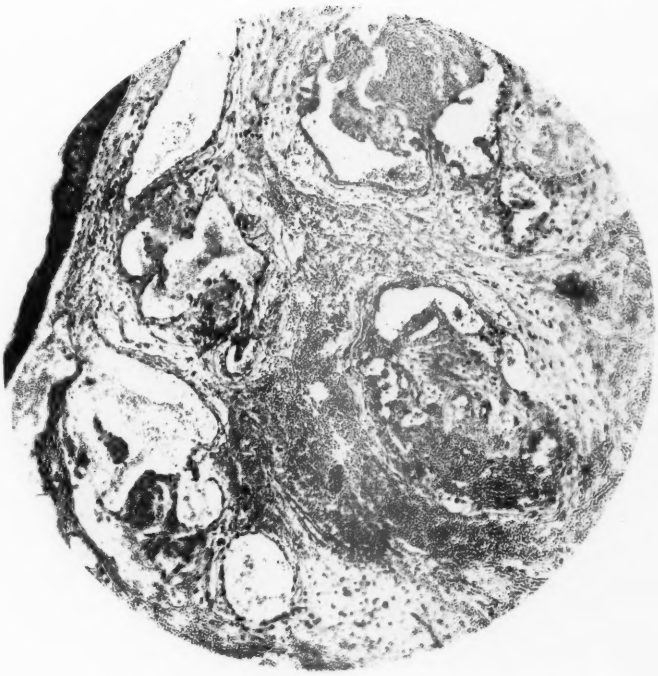

Fig. 1. Varix of vocal cord; organizing, fenestrating thrombi in blood spaces.

Fig. 2. Varix of vocal cord. Dilated vein occupying middle zone of papillary nodule with epithelium on both sides.

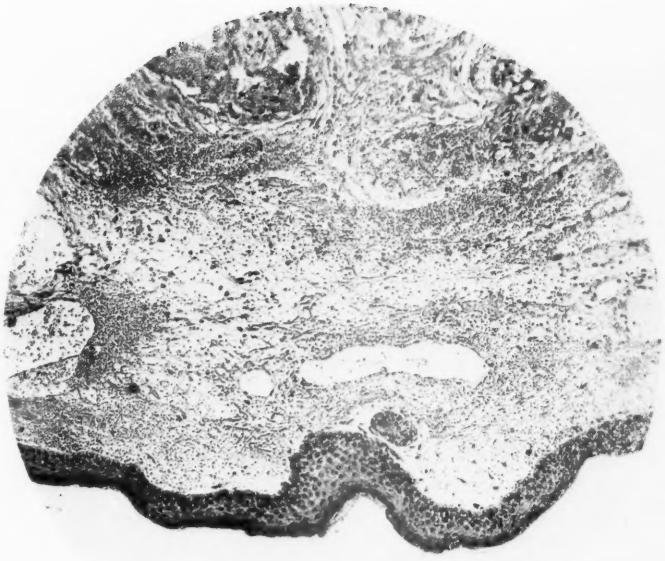

Fig. 3. Varix of vocal cord. Surface edema and capillary ectasia overlying thrombosis blood spaces.

done under local anesthesia, by direct laryngoscopy, with a cup forcep. No difficulty was encountered. The patient left the hospital the same afternoon, and has been seen at regular intervals up to the present time. The larynx appears entirely normal.

Laboratory report: "Papilloma of cord with large dilated blood spaces containing hyaline areas within their lumina, evidently fenestrated thrombi. There are perivascular areas of myxomatous degeneration. (See figures 1, 2 and 3.)

The specimen is essentially an angiomatous papilloma and might be called a hemorrhoid of the cord. It has not the same prognostic significance as the usual papilloma of the cord and will not recur."

A diligent search of the literature was made to see if this condition had previously been described. Faunce[1], in 1923, reported a similar condition. While he mentioned "hemorrhoid" as rather descriptive of the lesion, he rejected that terminology on account of its indelicacy when applied to the larynx. To us the word "varix" presents more clearly what was seen in the patient clinically and microscopically than any other terminology. The term "papilloma" brings to mind the idea of proliferation which is never present in this condition we are reporting. Even when the term "papilloma" is qualified as being "angiomatous" the implication of proliferation and possible return in the same area remains. We feel, therefore, that the designation "varix" is more appropriate than "angiomatous papilloma."

It is not our wish to appear to have rediscovered this condition. Rather, the purpose of this report is to re-define the condition and to draw the attention of the profession to a lesion which is of considerable importance in industrial areas. While it is barely possible that if the patient continues to work under the same conditions, a fresh lesion may appear in some other part of the cord, it is of inestimable value to be able to state to that patient that the lesion is a varicosity and to say definitely that it will not recur at the site of removal.

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XLIII

PROLONGED POST-OPERATIVE ANESTHESIA IN TONSILLECTOMY*

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AND
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For many years surgeons operating upon the tonsils have asked in vain for a substance which would increase the post-operative comfort of their patients. A great number of drugs have been suggested, tried and later discarded in an attempt to find the ideal. These preparations have been unsatisfactory largely for one of two reasons: either the technique was complicated, or the toxicity of the drug itself did not warrant its substitution for novocain.

Trotter[1] reports 126 tonsillectomies performed under quinine-ureahydrochloride and claims anesthesia was present for 24 hours or more. He reports no untoward results with this method giving a single injection into each side of the throat. We were unable to confirm his results, perhaps because of some fault in our technique.

In our attempt to discover a substance which would produce beneficial prolonged anesthesia for tonsillectomy, both nupercain,[2] .01 per cent and pontocain,[3] 0.1 per cent, were tried. A prolonged anesthesia was obtained, more marked with nupercain, but these preparations were abandoned because of the high degree of toxicity and the varying susceptibility to them.

A recent addition to the field of local anesthetics, eucupin, was brought to our attention. Eucupin dihydrochloride† with 1 per cent procain hydrochloride has been shown to give satisfactory prolonged surgical anesthesia in proctology. Eucupin, isoamylhydrocupreine, is an almost tasteless white powder in the quinine family. It is soluble in alcohol, ether, chloroform, hot vegetable and animal oils and fats, but insoluble in water. The dihydrochloride, which we use, is soluble in 15 parts of water, and easily soluble in alcohol.

*Department of Otolaryngology of Post-Graduate Hospital of Columbia University.

†Rare Chemical Company, Nepera Park, New York, supplied the necessary material for this experiment.

It is precipitated by alkalis, but has a marked germicidal action. Morgenroth and Bumbke[4] have shown it to kill streptococci in 1:4000 dilution, and staphylococcus aureus in 1:800 dilution. It is bacteriostatic to staphylococcus aureus in 1:160,000 dilution. The toxicity is extremely low, according to Kilbourne,[6] who established the minimum lethal dose at 150 mg. per kg. of body weight, which is one-tenth that of nupercain, though twice that of procain. The investigations of Dawson and Garside[7] demonstrated the extreme rarity of idiosyncrasy, though repeated injections may cause its development. In over 300 cases we have observed no evidence whatever of sensitivity. Other reports were of several cases of urticaria, which developed a few days after the injection of the eucupin-procain solution, but soon cleared up without treatment. As to the local irritation, I refer to Kilbourne's and De Tackac's[8] experiments, which showed that none of the injections of eucupin dihydrochloride in isotonic solution, caused any local irritations. Water solutions have been followed by sloughing. Solutions stronger than 0.2 per cent may cause local reactions, therefore higher concentration should not be used.

The action of eucupin in animals is similar to that of quinine, the difference being mainly quantitive. Large doses of eucupin stimulate the vagus center and the respiratory center of the medulla. On the smooth muscles it shows an inhibitory action and causes relaxation. Direct application of 0.1 of 1 per cent paralyzes the sensory nerve fibers.

The prolonged anesthetic action of eucupin is explained by Bylsma,[5] who analyzed tissues four days after injection and demonstrated the presence of the drug. The slow diffusibility in the tissues, he concluded, is the reason for the extended period of freedom from pain. Because of the slow absorption and consequently delayed initial action, procain hydrochloride 1 per cent was added to the solution.

We have used eucupin-procain solution as a local infiltration anesthetic in more than 300 tonsillectomies, both clinic and private, over a period of more than a year, with very satisfactory results. In a series of 25 patients we injected eucupin-procain solution in one side of the throat and procain 1 per cent solution in the other. This proved unsatisfactory—the patients could not tell which side had the more pain. Much better results were obtained when eucupin-procain solution was used on both sides of the throat. In the first 100 cases eucupin 0.1 per cent with procain 1 per cent was used. As no evidence of idiosyncrasy was observed, we used a solution of

eucupin 0.2 per cent and procain 1 per cent. More satisfactory results were obtained with this solution, which we used for the remainder of the series.

TECHNIQUE

(a) Because of the incompatibility with alkalies the syringe should not be boiled. If boiled, it should be thoroughly cleansed with sterile water, otherwise the eucupin may be precipitated.

(b) Because of the slow absorption of the drug, about five minutes should be allowed to elapse between injection and beginning of operation.

(c) The technique must be perfect.

(d) The amount used may be between 10 and 15 cc., although there is no contraindication to the use of a greater quantity. No more should be necessary.

(e) Because eucupin relaxes smooth muscle, eight drops 1:1000 epinephrine should be added to each 30 cc. of the solution.

POSTOPERATIVE COURSE

No case showed any evidence of systemic reaction, a phenomenon we occasionally encountered with procain alone. Postoperative hemorrhage was less in this series than in that with procain, and no evidence of local irritation of the tissues was noted. The healing process was unaffected.

Patients were tested for analgesia by one of us (H) at 2, 4, 6, 12 hours postoperative by touching the anterior pillar with a blunt probe. On some occasions a long tonsil needle was used. In those cases, in which the anesthesia was perfect at operation, the patient was absolutely free from pain for from five to twelve hours. In some patients a slight sensitiveness was present, increased by swallowing, but in others no discomfort was felt for 24 hours, in a few, for 48 hours.

Postoperative oozing, which occurred in some patients, naturally increased the sensitivity of the parts, and this was added to by the manipulations to check hemorrhage. Even in these patients, to some of whose fossæ pressure sponges and, on occasion, hemostatic forceps were applied, the analgesia was adequate to prevent discomfort.

In those cases in which anesthesia was not perfect at operation, the patients complained throughout the postoperative period. We consider this to be the fault of the technique rather than of the solution.

We do not consider that we have found the ideal agent for the sufficient prolongation of the postoperative anesthesia, but we feel that we have progressed far beyond the analgesic period possible with any local anesthetic in use today.

SUMMARY

1. Over 300 tonsillectomies under local infiltration anesthesia with eucupin-procain solution are reported.

2. No idiosyncrasis have been observed.

3. Anesthesia has been adequate.

4. Postoperative freedom from pain has been obtained from five to forty-eight hours.

5. No toxic symptoms have appeared.

6. No local tissue reaction has been encountered.

7. Healing has not been delayed.

8. In our opinion, eucupin-procain solution has proved itself superior both in anesthetic efficiency and in the prolongation of analgesia to any other local anesthetic in our experience.

124 EAST 84TH STREET.

303 EAST 20TH STREET.

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XLIV

STUDIES ON THE INTRANASAL PREVENTIVE TREATMENT OF POLIOMYELITIS*

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NEW YORK

In recent months there has been much interest in the zinc sulphate spray for the prevention of poliomyelitis. This has been particularly so since the publication of Peet, Echols and Richter[10] last June describing its application. As early as 1920, Flexner and Amoss[8] employed chloramin-T and dichloramin-T in an attempt to sterilize the nasal structures of the monkey. They concluded that antiseptic chemicals were of doubtful value. The recent interest in the employment of chemicals to prevent the entrance of the virus of poliomyelitis into the tissues of the central nervous system is based upon the work of several experimenters. Faber and Gebhardt[7] and Brodie and Elvidge[6] emphasized the fact that the nasal cavity is the most usual portal of entry of the infecting virus. Armstrong[1] was able to prevent infection by the intranasal route after he instilled picric acid solution into the monkeys' noses. Armstrong and Harrison,[2, 3, 4, 5] Schultz and Gebhardt[13, 14, 15] and Sabin, Olitzky and Cox[12] have shown repeatedly that intranasal inoculation with the virus could be prevented by the local use of various chemicals. On the basis of the work done by Armstrong and Harrisson, a picric acid-alum spray was used in the Alabama epidemic of 1934. It was generally agreed that the technique of application of the spray was faulty. Lennette and Hudson[9] demonstrated that the intravenous introduction of the virus was without effect when the olfactory tracts were severed.

The chemicals used by these experimenters included alum, tannic acid, picric acid, zinc sulphate and several dyestuffs. The effect is not directly on the virus, but rather upon the nerve filaments, in some way destroying their effectiveness as pathways for the transmission of the virus. The effect of the chemicals is not lasting, since most monkeys succumb in time to repeated instillations of the virus. The exact nature of the changes which account for this has

*Read before the Eastern Section of the American Laryngological, Rhinological and Otological Society, Philadelphia, January 7, 1938.

not been determined. The nasal mucous membranes of several monkeys treated with picric acid were studied microscopically by Lillie, working with Armstrong and Harrison.[8] He found that it was not possible to distinguish between the membranes of the treated and untreated animals.

In 1935 Armstrong and Harrison[2] reported that the introduction of a 4 per cent solution of alum into the noses of monkeys would prevent infection. Shortly thereafter Sabin, Olitzky and Cox found that the intranasal application of alum or tannic acid over a period of several days protected the monkey against subsequent infection by the nasal route. Instillations repeated daily for three days created a resistance which would last for some time. When this treatment was continued for five days or more, a majority of their animals (eleven out of fifteen) were still protected a month later, and seven of them two months later.

Schultz and Gebhardt[13] experimented with a long list of chemicals, forty in number. They found that the use of picric acid daily for several days would protect the nasal structures against the infecting virus. Later they concluded that zinc sulphate was deserving of a trial in man.[11] Two hundred and forty animals were tested with this chemical. They found that "two or three successive daily sprays with 1 per cent zinc sulphate will protect all or nearly all of the animals so treated against the virus administered, for one month after the treatments had been applied." Many of the animals in this experiment survived the repeated instillations of the virus for two and even three months. In one experiment eight monkeys received a single spray of zinc sulphate. They were given seven virus instillations from 25 to 32 days later. Seven of the eight survived. As a result of these studies Schultz and Gebhardt suggested the use of a solution of 1 per cent zinc sulphate, 0.5 per cent sodium chloride and 1 per cent pantocain. It must be remembered that the nasal passages of monkeys are much more spacious than those of humans.

Their suggestion was followed by Peet, Echols and Richter,[10] who advised that the spray must be applied directly to the olfactory area by means of a bulb atomizer employing a special spray tip. They did not feel that the introduction of the solution in the Proetz position would be as safe as the spray. They mentioned, however, that the Proetz position might be used when small children were uncooperative and the spray tip could not be introduced. According to them, the tip must be passed along the space between the middle turbinate and the septum (olfactory sulcus) and beyond the middle turbinate. If it comes in contact with the roof of the nose

it must be slightly withdrawn. One cubic centimeter (15 minims) of solution is required for each side. When the nasal passage is found to be occluded they advise preliminary shrinkage with ephedrine or benzedrine inhalant. They call attention to the fact that when the solution is properly applied the sense of smell is temporarily lost.

The writer was stimulated by the work of Peet and his associates to undertake studies to evaluate their method. Up to the present time nobody has taken into account the space available in the average nasal passage for the introduction of the nasal spray tip. An examination of the nasal passages of 157 children, other than those admitted for nasal infections, was carried out in the wards of the Kings County Hospital. The study had as its object an estimation of the amount of space each nasal passage afforded for the introduction of the tip. A preliminary study was carried out to determine the amount of space which would be required both in the nose, which was subjected to preliminary shrinkage, and that which was not so treated.

The nasal passages were classified as follows:

1. Sufficient when the space was enough to permit the passage of the tip beyond the middle turbinate, with or without shrinkage.

2. Insufficient when there was some, but not enough space for passage of the tip, even after preliminary shrinkage.

3. None when there was no space available, even after shrinkage.

In all our experiments a solution of ½% cocain hydrochloride and 1:5000 adrenalin chloride was used for preliminary shrinkage. All of our experiments were done on one side of the nose. Following are the results of this study:

Analysis of 314 nasal passages of 157 children, ages 2 to 16 years:

	Passages
1. Sufficient space for passage of tip:	
One side of nose	46
Both sides of nose (15)	30
	76
2. Insufficient space for passage of tip:	
One side of nose	67
Both sides of nose (14)	28
	95

3. No space for passage of tip:	Passages
One side of nose	63
Both sides of nose (40)	80
	143

Of 157 children, 15 (10%) had enough space in both sides of the nose to permit the use of the nasal tip; while 46 (29%) had sufficient space in only one side. Of 314 passages, 238 (75%) had insufficient or no space available for the introduction of the nasal spray tip.

The most frequent cause of an obstructed passageway was a high convexity due to deviation or deflection of the septum on one or both sides. Occasionally the convexity was found on a level lower than that of the middle turbinate, so that the region of the olfactory sulcus could not be seen. In a good many children the septum was neither deflected nor deviated, but the nose was so narrow that the structures were crowded.

An attempt was made to determine the percentage of children who would co-operate in carrying out this procedure. A child was considered as uncc-operative if, after three successive attempts, we could not induce him to submit willingly to the introduction of the nasal tip. It was found that only two or three out of each group of ten would permit the introduction of the tip. The use of a local anesthetic did not influence this proportion. It should be taken into account, however, that this lack of co-operation might not exist during an epidemic, when the assistance of the parents is available.

At the suggestion of Dr. Imperatori, examination of the passages of children recently ill with poliomyelitis was undertaken. Twenty children were studied. Their nasal passages were in no way different than those of children unaffected.

Studies were made to determine the value of the instillation of the chemical by means of a spray or dropper, with the patient in the dorsal recumbent position and his head hanging over the end of the table, so that the top of the head was parallel to the floor. Skiodan and Thorotrast were used after a preliminary shrinking spray. After the introduction of 1 cc. of the opaque solution the head was kept in position for three minutes, following which a roentgenogram was taken. The opaque substance could be demonstrated in the olfactory area on each film, regardless of the amount of space between the middle turbinate and the septum. Some of it

could be seen going toward the pharynx. The dorsal recumbent position, with the head in complete extension, so that the top of the head is parallel to the floor, meets with no objection from the children. It is quickly assumed and offers no difficulty. During the recent Toronto epidemic Pentecost used a somewhat similar position. He reported success with all of his subjects when he used a special catheter for introducing the medication. We have found that a special introducing instrument is unnecessary; that an ordinary medicine dropper gives 100 per cent successful applications.

A large group of adults, and a few children, were x-rayed immediately after Skiodan was sprayed into the olfactory area, according to the technique suggested by Peet. This was done after preliminary shrinkage. In all but three films the opaque substance was found on the floor of the nose, passing into the pharynx.

We were able to observe the effects of the zinc sulphate pantocain spray in four adults. Two principal reactions were noted: The loss of the sense of smell and headache. The sense of smell was lost within a few minutes after the application, as was demonstrated by the olfactometer. The inability to smell lasted eighteen days in two patients, five days in another, while the fourth was lost sight of after the fifth day, at which time she could not smell. The headache usually came on within two or three hours after the use of the spray. It was so severe in three of the subjects that opiates were required for relief. One of the patients experienced headache for several days; a second had severe headache all of the first night; the third suffered for eight hours, while the fourth had headache for only one hour. Because of these reactions we could not get additional volunteers for further trial with the zinc sulphate spray. Despite this small number of subjects, it is plain that headache must be taken into account in this procedure and that severe headache may occur. Pentecost[11] found that headache followed in every case and persisted for from two to six hours. It was more severe in patients over 12 years of age. The report of the Toronto experience,[16] based upon the use of the zinc sulphate spray in 4,713 children during the recent epidemic, did not stress this symptom, yet one is impressed with the fact that headache was experienced by many of their subjects and that in some it was quite severe.

A group of 4,713 children between the ages of 3 and 10 years received the zinc sulphate prophylactic spray in Toronto. Most of them suffered temporary discomfort, while some complained of severe pain between the eyes which continued for several hours. The

symptoms noted were headache, nausea or vomiting and stiffness of the muscles of the neck. Elevation of the temperature was commonly noted. Of 859 children tested for their sense of smell, 152 (17.7%) developed anosmia. It was concluded that serious limitations are placed upon the use of this spray as a public health procedure because skillful otolaryngologists are required. They called attention to the fact that children were so disquieted by the procedure that it was with difficulty they were brought back to the clinic, ten days later. The Toronto group felt that the nasal spray technique was not successful. They gave no reason for this. The main reason lies in the fact that they did not take into consideration that only a relatively small percentage of nasal passages (25% according to this study) will permit a satisfactory spraying of the olfactory area with the special spray tip.

The reaction occasioned by the zinc sulphate led to the consideration of other chemicals which might be less irritating. Armstrong and Harrison and others had tried picric acid and found it a good virucidal agent. The first authors mentioned that the 1 per cent solution did not cause severe local reaction when they tried it on themselves. This chemical was therefore tried on a small group. After preliminary shrinkage, a 1 per cent aqueous solution of picric acid was introduced into one side of the nose, by means of a medicine dropper, in eight adults. This was done in the dorsal recumbent position with the head extended over the end of the table so that the top of the head was parallel with the floor. A reaction which was common to all consisted of severe smarting within 15 to 30 seconds after the drops were introduced. There was lacrimation of slight to severe degree. All had a sense of stuffiness in that side of the nose. With few exceptions the reaction was completely spent within a few minutes. In one of the subjects a transient but severe conjunctivitis occurred. This was thought to be due to the fact that he blew his nose and forced some of the picric acid up through the nasolacrimal passage. Headache lasted as follows, in eight subjects: None in three; in the remaining five it lasted five, ten and fifteen minutes, and one hour, and one hour and twenty minutes. Among these eight was a member of the house staff whose headache of one hour's duration was much milder than the headache of six hours' duration he experienced on a previous occasion when treated with the zinc sulphate. Complete loss of the sense of smell was revealed by the olfactometer ten minutes after the drops were introduced, in each case. This anosmia was still present on the fourteenth day in two cases. In two others it lasted five days, while in the remaining four it was present for an average of three days.

SUMMARY

1. The introduction of the nasal spray tip into the olfactory sulcus of the noses of children is difficult or impossible because:

a) A great majority of the nasal passages do not afford a sufficient space, even after shrinkage, to permit the passage of the spray tip between the middle turbinate and the septum. Two hundred and thirty-eight out of three hundred and fourteen nasal passages (75%) were found to have insufficient or no space available for passage of the tip.

b) A majority of the children are afraid of the nasal spray tip and will not submit willingly to its use. This may not be so, however, when the children are impressed with the importance of what is being done, by their parents.

2. Our limited study, added to the extensive experience of the Toronto group, establishes the fact that some headache occurs in all, while very severe headache occurs in some who are subjected to the zinc sulphate spray.

3. The olfactometer should be used to check the effect of the chemical application. If the solution is applied to the olfactory area the sense of smell is lost; if the sense of smell is not lost the olfactory area has not been properly covered.

4. As a result of our x-ray studies and those in which picric acid was used, we suggest the following as the simplest and most effective method for applying the solution: After preliminary shrinkage with cocain hydrochloride ½% and adrenalin chloride 1:5000 solution, the patient is placed in the dorsal recumbent position with the head extended over the end of the table so that the top of the head is parallel to the floor. The solution is introduced by means of a medicine dropper and the head is kept in position for three minutes. This technique gives uniformly good results and eliminates the necessary consideration, when the spray is used, of the available space in the nasal passage and the co-operation of the subject. The objection that the child will swallow the solution when it is introduced in this manner applies equally well to the spray. This can be seen by studying the films taken after opaque substances were used with both methods. Since the zinc sulphate spray contains only three-tenths of a grain of pantocain in the 30 minims used, a toxic dose could not be swallowed. Inasmuch as pantocain is incompatible with picric acid, it cannot be used with this chemical.

5. Picric acid in 1 per cent aqueous solution is suggested as a prophylactic intranasal application for poliomyelitis. The three-

tenth grain used does not constitute a toxic dose. It is less objectionable than zinc sulphate because it causes relatively little discomfort. The olfactometer tests indicate that its effect may last longer than fifteen days after a single application. A technique which involves several initial, successive applications, and later periodic ones, will very likely give protection which will last through an epidemic or during a period in which the disease is prevalent. It should be tried on this basis.

A larger group of children can readily be treated with the extended head position. They will co-operate by assuming this position, whereas they might not permit the introduction of the spray tip.

CONCLUSION

As a result of the above studies, picric acid in 1 per cent solution deserves a trial as a preventive nasal instillation for use during epidemics of poliomyelitis, or when this disease is prevalent. More extensive trial should be given to establish its value on a firm basis.

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Society Proceedings

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Meeting of Monday, December 6, 1937

THE PRESIDENT, DR. WALTER H. THEOBOLD, IN THE CHAIR

The Otolaryngological Manifestations of Hemophilia

CARROLL L. BIRCH (by invitation)

(Author's Abstract)

Hemophilia is usually classified under the hemorrhagic diatheses. It is characterized by habitual hemorrhage from various parts of the body, which may be spontaneous or may follow injury. The hemorrhage is excessive and out of all proportion to the extent of the injury. The hemorrhagic manifestations usually begin early in life. In the writer's experience with 107 cases of hemophilia, 30 per cent had hemorrhages within the first three weeks of life; 85 per cent had suffered hemorrhages within the first three years.

This disease is hereditary and is sex-linked in its transmission. Males only have the disease, but it is transmitted by the unaffected female. All the daughters of a hemophiliac become transmitters of the defect. Theoretically, 50 per cent of the daughters become transmitters like themselves. Hemophilia is never transmitted through the normal male and is never passed directly from father to son. The sons of hemophiliacs are normal and all their descendants are normal.

The only constant laboratory finding is prolongation of the coagulation time of the blood. In general the longer the coagulation time the more severe the hemophilia.

Hemorrhage may occur any place in the body, subcutaneously, intramuscularly, nasally, from the gums, throat, stomach or intestine. A very common site for hemorrhage is the kidney, while the most characteristic location is into the joints. More rarely hemorrhage may arise from the lungs or into the spinal cord or brain resulting in paralysis. All the 107 patients observed had subcutaneous hemorrhages, and all but one had intramuscular hemorrhages. Hemarthroses were found in 85 per cent of the patients. Of the 16 patients

who had suffered no joint hemorrhages, only 6 were beyond the age of 12 years. Permanent joint deformities were found in 68 cases. Of the 39 who showed no crippling, only nine were older than 12 years. In the milder cases, permanent crippling is the result of repeated hemorrhages into the same joint, which frequently occurs during the second or even the third decade of life. With these facts in mind it is clear that the percentage of hemarthrosis and deformity appears to be lower than is actually the case. The distribution of other hemorrhages was as follows: Forty-two patients had kidney hemorrhages; 37 had peritoneal, gastric, or intestinal hemorrhages; 101 had nasal hemorrhages; 98 had hemorrhages from the mouth, including teeth, gums and tongue; six had brain or spinal cord hemorrhages; and five had lung or pleural hemorrhages.

Subcutaneous and intramuscular hemorrhages assume serious proportions in dangerous areas such as the head and neck. Many of these are traumatic as from biting the tongue or inside of the cheek. Bleeding from the gums may be caused by the eruption or loss of the baby teeth, by foreign or hard particles in food, as fish bones or nuts, the bristles from a hard tooth brush, etc. In early childhood biting on hard sharp toys is a frequent cause of serious mouth hemorrhage. Falling with a pencil or other article in the mouth is not a rare cause of fulminating loss of blood. Infections, pyorrhea or acute tonsillitis, are often accompanied by extensive hemorrhage into the floor of the mouth, into the tongue and extending down into the neck. Sometimes bleeding into tissues of the throat and neck becomes so extensive that swallowing is impossible and breathing difficult. Other causes for hemorrhage into this area are abscessed and impacted teeth.

These patients present a pathetic picture. The swelling and discoloration extends into both eyes; the lids become so swollen that the eyes cannot be opened, the tongue so enlarged that the mouth is held open, the saliva cannot be swallowed and drools from the corners of the mouth. Although it has seemed at times that the pressure must be relieved, we have always refrained from surgical interference and to date all of these patients have recovered.

Epistaxis is almost universal in hemophilia. It may take the form of small frequently recurring hemorrhages or the patient may have severe depleting loss of blood. In seven of our patients epistaxis was the earliest recalled hemorrhage.

In 22 patients the initial hemorrhage occurred from the mouth (including tongue, lips and gums). Thus approximately 30 per cent of hemophiliacs have initial symptoms which would direct them first

to the ear, nose and throat specialist or the dentist. When it is remembered how serious surgical interference is in this disease the responsibility of the first consultant becomes apparent.

In a survey of 113 deaths in hemophilia 57 per cent died before they reached the fifth year of life. Twenty-five per cent of these deaths were caused by surgical procedures; 15 per cent were the result of circumcision; 6 per cent followed tooth extraction, although the diagnosis was known in each instance before the tooth was extracted; one resulted from tonsillectomy; one from a lanced throat, the throat having been lanced to relieve pressure symptoms of hemorrhage; one was the result of a lanced hematoma of the scalp; and one followed vaccination. This is the only incidence of hemorrhage following vaccination, even though all but one of our patients over seven years of age have been vaccinated. Small accidental cuts were fatal in 23, six had fatal epistaxis. There were three fatal hemorrhages from the throat during the course of diphtheria. One bled to death from the gums while cutting a baby tooth. Two patients died of natural causes, while the remainder died from hemorrhages elsewhere in the body.

From this brief survey it is plain that the hemophiliac is subject to frequent and severe hemorrhages in the region of the head and neck and that otolaryngologists have a definite responsibility to these patients.

DISCUSSION

Dr. Katsuji Kato (by invitation): Dr. Theobald was good enough to invite me to discuss this exceedingly instructive paper. I express my hesitancy in accepting the invitation for the reason that my experience with hemophilia is very limited, and I decided to attend the meeting in order to learn rather than to discuss.

This splendid presentation by Dr. Birch is extremely interesting. I have never before seen such a complete group of pictures illustrating the various manifestations of this important disease. Since I am interested primarily in various types of blood diseases in infants and children, I may speak of one or two matters which are uppermost in my mind tonight.

Theoretically, pediatricians should encounter many cases of hemophilia, since the bleeding tendencies of hemophiliacs are more apt to be manifest in childhood because of a greater possibility for trauma in this period of life than in adult life. In my personal experience, however, the incidence of the disease is low. This may be due to the fact that we missed the diagnosis, not so much because of our

lack of interest in the disease but rather because of the lack of a simple method of estimating the coagulation time of the blood, easily applicable to infants and children. I suppose the most reliable method, either that of Lee and White or that of Howell, is to use venous blood, but venipunctures in small patients are not as readily practicable as in adults. To be sure venous blood can be obtained by the puncture of the superior longitudinal sinus or of the external jugular vein in infants, but this is practised only in exceptional cases.

In routine work, however, capillary blood must be used. I have been for some time using a capillary blood coagulation test, in spite of the objections often raised. I use a hanging drop slide with a central depression, about 15 mm. in diameter and 3 mm. in depth. Having coated the surface of this depression with a small amount of melted paraffin, I take the slide to the bedside. From a clean deep puncture wound of either a finger or an ear lobe, I allow two or three drops of blood to fall into the paraffin-coated depression and immediately place a small clean glass bead (3 mm. in diameter) and after covering with another slide and closing the depression cavity with vaselin, I tilt the slide from time to time. When the glass bead becomes immovable upon tilting the slide in vertical position, the coagulation is regarded as complete. I remember that Dr. Haden stated that it is a waste of time to try to determine coagulation time with capillary blood, but for the reason stated above I use this method routinely. Whenever an abnormally prolonged coagulation time is obtained, a check of course must be made on venous blood. For routine purpose, however, I find this capillary test to be fairly satisfactory.

Another matter of tremendous importance to us is the fact that we do not as yet know the exact mechanism of delayed coagulation in hemophilia. The problem has been investigated recently by Bendien and van Creveld (1937) of Holland. They precipitated a certain fraction of globulin from the normal blood serum and noted that this fraction of the protein (called coagulation globulin), when administered intramuscularly or otherwise, will reduce the coagulation time of hemophiliac blood. I am not in a position to discuss the chemistry of their investigation, but it seems to offer a great promise in the future studies of the nature of coagulation mechanism. Dr. Birch in 1933 aroused a great deal of interest in the treatment of hemophiliacs by the use of female sex hormone, which has subsequently been tried by a number of observers, some with good and others with poor results. To whatever theory of coagulation we adhere, we should not cease to search for the real factor which char-

acterizes the defect in the hemophilic blood. I think the hemophiliacs can never be permanently cured, but by the proper use of remedies we can more or less correct the abnormalities of hemophilic blood or at least keep the coagulation time of the patients to the minimal level.

DR. T. C. GALLOWAY: This was an amazing number of cases and I would like to know from how large a clinical group they were collected. We have all seen many cases that came in as bleeders and we have found on careful study that they were not hemophiliacs. They turned out to be cases of purpura, jaundice, and so on, with prolonged bleeding time but not hemophilia. I have seen one case that was an undoubted hemophiliac and there was severe bleeding from an antrum puncture. This man, a doctor, knew he was a hemophiliac and he had had previously a severe hemorrhage following a slight injury. Packs and various other measures failed, but the bleeding was controlled without difficulty by dessicating diathermy. I wonder whether Dr. Birch has tried this.

DR. FRANCIS L. LEDERER: Dr. Birch's time was very limited for a complete presentation of this subject, but here is a person who can make romance out of a blood dyscrasia. I am sorry she did not have time to tell us more about the ulcerative lesions about the pharynx which accompany other blood disturbances. The largest percentage of these cases come from the nose and throat department. We encounter numerous lesions about the pharynx, particularly those occurring with purpuras and leukemias, which she might have discussed had time permitted.

The point Dr. Kato made about the tonsil coagulation and the lack of dependability of the ordinary methods employed is important. I have the feeling that this is a useless procedure. We routinely do these coagulation tests to satisfy the laity and they may or may not mean anything. First of all is the question of technique, and secondly, the fact that there is a quiescent interval in hemophilia during which time the test is normal. Dr. Birch might tell us whether a hemophiliac would at all times show a positive test.

When considering the number of cases, Dr. Birch is fortunate or unfortunate in having a reputation in this field, and many cases are sent directly to her from all over the country. I believe that in Osler's entire experience he saw but sixty cases. Dr. Birch has gone beyond the clinical scope, for we know she has been abroad and interrogated members of the royal families and investigated and studied the family tree in regard to hemophilia. Such a discussion may be of considerable interest to those present.

We should refrain from all vigorous local efforts to control a hemorrhage. Following Dr. Birch's experience we might just as well try the "hands off" policy, giving transfusions as a supportive measure. The fact that in Dr. Galloway's case the hemorrhage stopped following diathermy, leads me to believe there must have been other factors present that played a role. Dr. Birch makes the point very emphatic, and it is well taken, that we should not disturb these lesions. We should expectantly wait for the clots to come away instead of trying packs or any of the local procedures usually employed in controlling hemorrhage.

DR. IRVING I. MUSKAT: It has come to my attention in my reading that some good effects have resulted from the local application of mothers' milk. I should also like to know if Dr. Birch has had any experience with estrogenic substances in hemophilia.

DR. FRANK J. NOVAK: Will Dr. Birch give us her experience with the use of the sex hormone and with extremely large doses of viosterol in hemophilia

DR. AUSTIN A. HAYDEN: I am interested in the minister who was attracted by the chiropractor's wife. Evidently this gentleman was old enough to have cut his teeth and to have lost several teeth, and I wonder why bleeding did not occur at that time.

Referring to the question that has been suggested in the discussion, is a hemophiliac always a hemophiliac, or are there times when he is not a hemophiliac? The questions that have been raised regarding coagulation time are very suggestive and I wish to hear what Dr. Birch has to say about the blood clotting methods commonly in use.

I must disagree with Dr. Birch about the fact that nose and throat men may not recognize these cases. I have heard them accused of a great many things, but I think any nose and throat man would know a hemophiliac if he had operated upon him, and the number, so far as I know, is very small.

DR. ALFRED LEWY: I might report a case of hemophilia that I was unfortunate enough to see. The patient was a young man, aged 21, with a history of bloody effusion into the knee joints. His brother had had the same condition and his uncle on the maternal side had died of a hemorrhage due to hemophilia. When I saw this young man he had what was thought to be a peritonsillar abscess. His temperature was 102 F., he had pain and dysphagia due to the obstruction for two or three days. There was already infiltration

under the tongue. I hospitalized him without making any incision because I was familiar with his history. During the night breathing became so obstructed that a tracheotomy had to be done. This was done through about two inches of infiltrated tissue, with the patient in a semi-recumbent position. Every bleeder that could be recognized was picked up and tied, the tube was inserted, but he continued to ooze from the wound. He died within twenty-four hours, I do not know whether of sepsis or from the bleeding. The whole front of his body was more or less covered with blood during the night, but apparently he did not die of loss of blood alone.

I wish to ask if Dr. Birch has any experience with the use of moccasin and fer-de-lance venom recently recommended locally for the control of such bleeding?

DR. G. H. SCOTT: Repeated transfusions were found to be of great value in one case of hemophilia.

The case was a recognized hemophiliac, aged 24, in whom the extraction of several teeth was deemed imperative.

Blood studies were made and several suitable donors were procured beforehand.

Following the extractions, the patient oozed steadily from the gums as had been anticipated. It was found that a transfusion would check the oozing for a more or less definite period, the length of time being quite similar in each instance. Frequent transfusions were then spaced so that each consecutive one was given before the clotting effect of the one preceding had worn off. The method proved successful in this instance.

DR. CARROLL L. BIRCH (closing): I would say concerning methods of determining coagulation time, that in hemophilia venous blood should be used instead of capillary blood, for the reason that the admixture of any tissue juice will shorten the coagulation time. I made quite a long series of determinations, using capillary blood and venous blood. The differences were very great and there seemed to be no definite correlation between the two methods. The clotting time of the venous blood was always longer. In small children we use a 27 gauge needle and a small syringe, and in most instances take the blood from the antecubital vein. We use Horoell's method of determining the coagulation time. We take 2 cc. of blood from patients under seven years of age and 5 cc. from the older ones. Only blood obtained by clean venapuncture without bubbles should be used. After the vein is entered the tourniquet should be removed to prevent stasis. It is a rather simple procedure, but yet every slip

in technique tends to shorten the clotting time. The coagulation time in the same hemophiliac varies from time to time. Many of them have cyclic variations. In most instances we have obtained our longest coagulation times in April and November, and our most serious spontaneous hemorrhages have occurred in the spring and fall. In the mild cases of hemophilia you may have a normal clotting time on one occasion and the next week find it considerably prolonged. They too pass through these cycles. In doubtful cases several determinations should be made at least a week apart.

We have never been able to substantiate the diagnosis of hemophilia in any of the women referred with that diagnosis. Most of them were chronic purpuras. The cases we have had have come from all over the United States, twenty-five were from Chicago, thirty-six from Illinois, the rest from outside the state. Many patients have been referred to us with the diagnosis of hemophilia who actually had purpura, scurvy, leukemia, etc.

We have used snake venom and in some cases it works very well. Used locally after tooth extraction it has been of some help. In the average case transfusion will control hemorrhage for forty-eight hours, but sometimes not so long. We have a boy in the hospital now who had an automobile accident, sustaining a fracture of the left femur, two lacerations of the scalp, a deep cut into the palm, a large hematoma of the chin, a small cut inside the mouth and a hematoma of the right knee. He has been in the hospital now for thirty-six days and has received fifteen transfusions.

As for electrocoagulation, we have used it in open wounds. We have not had any help in intramuscular hemorrhages, but for hemorrhage due to a cut, where blood is being lost outside the body, we have used the coagulating needle with some success.

We have obtained results with whole ovarian extract but not with theelin. Some have obtained the same results from placental extract. In England they claim results with extract of white of egg. As Dr. Kato said, it is probably a split protein. Saliva, pineapple juice, trypsin and all other proteolytic enzymes will reduce the time to normal. It is probably a proteolytic enzyme which produces this result, but just what it is no one really knows as yet.

We have used mother's milk, and it probably is the same substance, an enzyme or a split protein. All of these things will clot the blood outside the body, but when applied to a bleeding point the clot forms but the blood wells up from beneath and washes the clot away.

The Clinical Application of the Galvanic Falling Reaction

EDWIN J. BLONDER

(Presentation published in full in this issue)

DISCUSSION

DR. LEWIS J. POLLOCK (by invitation): Since the apparent correlation between disassociated function of the semicircular canals and specific localization of lesions in the brain stem has not been confirmed, it is important to study other methods of examination of function of the vestibular nerve.

There is some controversy concerning the site of action of electrical stimulation in producing galvanic falling. Some believe that the current stimulates the end organ, others the vestibular nerve, still others the brain stem. There is also some difference of opinion as to whether the otolith organ alone is stimulated or that the stimulus acts as does rotational and caloric stimulation of the semicircular canals. It may be of interest to point out some of the differences between response in muscle and nerve and the vestibular apparatus. In muscle and nerve the response to electrical stimulation occurs only in the make or break of the current and not during the continued flow except under certain conditions when an initial tetanus of short duration is produced. The vestibular response, on the other hand, requires the continued passage of a direct current and persists as long as the current flows. In muscle and nerve, responses occur not only to direct current but to alternating current, if the frequency is not too great. There is no response of the vestibular apparatus to alternating current.

It is well known that an adequate stimulus of muscle and nerve must not only be of sufficient strength but must last for a certain minimal time. When we determine the shortest duration of a stimulus which is effective at twice the voltage necessary to produce an effect at infinite duration, we obtain the chronaxia of a tissue. The supposed chronaxia of the vestibular nerve is known to be as much as forty times that of spinal nerve or muscle. This suggests that, since the responses of the labyrinth are quickly effective to slight changes of position of the head, we are dealing not with a chronaxia of a tissue but a physical change, perhaps in the endolymph comparable to a change produced by rotation and caloric change.

When a mixed nerve is stimulated response occurs in both motor and sensory functions. Although galvanic nystagmus is noted,

it is of an entirely different order from that obtained by turning and is a lesser part of the response. Finally a falling is produced by repetitive stimuli at a frequency far greater than is the response of a nerve or muscle, although asynchronous stimulation of many fibers may explain this. It would seem that the response of falling to galvanic stimulation is the result of changes produced in the end organ or the endolymph or both. There is an indication for further work both upon the clinical application of this method and its physiology.

DR. SHERMAN L. SHAPIRO: This paper represents an attempt at exploring a method of vestibular examination which has long been known but has had little clinical application. The author is to be commended for this reason as well as for his refinement in technique of applying the test.

Dr. Blonder's clinical material represents a large variety of cases. I do not know that he claimed any special value for the falling reaction as compared with galvanic nystagmus except the ease of application. This claim, however, is not true if the Frenzel glasses are used when the test for galvanic nystagmus is greatly facilitated. I have been able to obtain this reaction in normal individuals with amounts of current as low as ½ m. a.

Another point to be cleared up is the value of Dr. Blonder's test as compared with the caloric reaction. I was interested in hearing the author mention two cases in which there was no reaction by the caloric method and in which, nevertheless, falling occurred towards the anode. The occurrence of such instances indicates a possibility of obtaining something in a diagnostic way from the galvanic examination that we cannot obtain by the caloric test.

As to the theoretical phase, first I wish to agree with Dr. Pollock that the make or break is not the important factor in producing a galvanic reaction. Anyone, for instance, who performs the test for galvanic nystagmus will note that it takes a little while for the eye movements to appear even when observed under the brilliantly illuminated Frenzel glasses. I do not know about the otoliths being the site of stimulation. Everything from the internal ear to the vestibular nuclei has been cast in this role by various authors. I can only state that in my own experience I have produced a good galvanic nystagmus, using larger amounts of current than ordinarily, in a patient whose eighth nerve had been sectioned intracranially and who had a dead ear by all usual criteria.

I did not hear Dr. Blonder state the level at which the cerebrospinal axis was severed in his cats. It is rather astonishing that no reaction was obtained, since the Utrecht school demonstrated years ago that the eye reflexes can be obtained even if only the abducens nucleus is still below the level of the section. It is now well established that neither the eye, body or extremity reflexes are dependent upon integrity of the cerebrum for their occurrence.

Dr. Ellison L. Ross: Dr. Blonder is to be congratulated on his careful and extensive work. It tends to place his method of testing on a par with the caloric and rotation tests that are more commonly used. It is a question whether the utriculus or the semicircular canals or the entire labyrinth is stimulated by Dr. Blonder.

Dr. Edwin J. Blonder (closing): I wish to thank Dr. Pollock, Dr. Shapiro and Dr. Ross for their discussion.

Dr. Pollock in his discussion has brought out much interesting and important information regarding galvanic stimulation.

With regard to Dr. Shapiro's statement regarding galvanic nystagmus, I have no controversy with those who can interpret galvanic nystagmus with such accuracy that it can be of definite clinical value. The literature contains many articles in which the authors have considered galvanic nystagmus as inconsistent and inaccurate for purposes of diagnosis, and I have quoted a number of these in my bibliography. I hesitate to place the same reliance on galvanic nystagmus as I would on caloric nystagmus or galvanic falling. We have found galvanic falling as dependable as the caloric stimulation.

Galvanic nystagmus and galvanic falling do not appear to be results of stimulation of the same portion of the vestibular system. Magnus and DeKleyn first demonstrated that when an animal with decerebrate rigidity is placed on his back with the nose forty-five degrees above the horizontal, the extensor tonus is increased. This increase in extensor tonus they localized to the otolithic apparatus. Pollock and Davis confirmed this with animals decerebrated by the anemic method. Dr. Loyal Davis and I produced an animal with decerebrate rigidity similar to those produced by Magnus and DeKleyn. We varied this extensor tonus by galvanic stimulation of the auditory bulb, thereby altering the influence of the otolith apparatus as considered by Magnus and DeKleyn and others.

There is no question but that galvanic nystagmus can be produced by stimulation of the eighth nerve. Wilson and Pike and

Neumann were so quoted in this article. We feel that small amounts of current stimulate the peripheral apparatus.

Regarding the question of the level of the decerebration, our preparations were not surgical decerebrations as performed by Magnus and DeKleyn. Pollock and Davis developed an ingenius method of producing anemic decerebrations at levels they desired by ligating the basilar and carotid arteries, which they checked by the use of dyes. The level of decerebration was determined by varying the point of ligation of the basilar artery. We included the sixth nerve in our preparations.

Dr. Ross brought up the question of whether a portion or all the labyrinth was stimulated by galvanic current. From our work it appears that the portion of the vestibular system concerned with posture is more responsive to galvanic stimulation, when a balance board is utilized.

Acute Bulbar Poliomyelitis Two Weeks After Tonsillectomy

ALICE K. HALL.

(Author's Abstract)

This case is reported in order to suggest the possibility of acute bulbar poliomyelitis in a differential diagnosis of paralysis of the soft palate, and to call attention to the fact that a patient with this type of a disease may first consult an otolaryngologist. It illustrates also the possible mildness of the systemic manifestations, even during the acute paralytic phase of the disease.

On October 30, 1937, Richard A., age 8, was examined at the Northwestern University Medical School, his complaint being a speech defect of two days' duration associated with regurgitation of liquids into the nose. His tonsils had been removed two weeks before and he had been attending school for the week prior to this examination. The striking finding was an almost complete paralysis of the soft palate, associated with stiffness of the neck and spine. A presumptive diagnosis of bulbar poliomyelitis was made and the patient was transferred immediately to the Chicago Municipal Contagious Disease Hospital where he remained for the usual twenty-one days. He returned home on November 18th and according to the latest report received two days ago, was up and about the house, having no difficulty in swallowing and only a slight speech defect.

The occurence of this infection twelve days after a tonsillectomy suggests a consideration of the possibility of surgical poliomyelitis comparable to surgical scarlet fever as a clinical entity.

Comparative Audiometer Tests

AUSTIN A. HAYDEN

(Author's Abstract)

As unnecessary "minimum requirements" increase the cost and thereby, to some extent, limit the use of any apparatus designed for wide clinical use, it is desirable to determine the value of such requirements. In audiometers pure wave form, which does add greatly to construction costs, has been considered necessary, especially by laboratory workers. To determine its real value in actual practice, thirty moderately hard of hearing people were tested with four audiometers. Three of these X (Sonotone), Z (Western Electric 6A and 2A), probably meet requirements of the Council on Physical Therapy, while Y (Marvel) has an admittedly imperfect wave form. Western Electric 2 A was arbitrarily taken as a standard for comparison because of its long established (but unfortunately far from sufficient) use and the literature it has created.

The four audiometers were placed in a small inside room of an otologist's office. The doors and windows in this and adjoining outside rooms were closed during the tests. The average noise level was then reduced from fifty to twenty db. A thick rubber hood around the air receiver with a similar cup over the opposite ear further lowered the noise level but to exactly what extent was not determined.

Audiometer X is provided with dials for octave (128 to 8192) as well as sweep frequencies (2500 to 17,000). Intensity is calibrated in steps of five decibels. It is provided with an automatic self-compensating pointer. Audiometer Y has octave, octave letter and sweep frequency range from 64 to 8192 with attenuation from 0 to 100 dbs. at all frequencies. Audiometer Z has two dials, one for sweep frequency marked with eight indicated points from 128 to 9847. For bone conduction tests, a ring is placed over the intensity dial for recalibration.

When being tested, the patient responded by the usual finger or electric signal. The average of a minimum of three readings by air conduction at each of the octave frequencies was taken to be the actual hearing loss and charted as such on the individual audiograms and recorded. These three readings almost never varied from each other by more than plus or minus five db. The tests were made by three experienced observers. Rest periods were used as needed to minimize fatigue-confusion of both patient and observer.

Fourteen of the thirty were found to have hearing losses greater than the Western Electric 2A could measure. For that reason their audiograms were discarded. The hearing losses of the remaining sixteen, as shown by each of the four audiometers, were averaged and plotted. The average difference over all frequencies is readily compiled and is not great.

SUMMARY

1. Four audiometers of different makes were investigated to determine their comparative performances for air conduction tests and the need for pure wave form in clinical practice.

2. Audiometer X consistently showed the highest hearing loss; Y showed a medium, but more erratic loss; and Z the smallest hearing loss. As compared with the 2A, the average differences were five db. plus for X, minus three db. for Y, and minus six db. for Z.

3. Audiometers X, Y and Z were capable of measuring greater hearing losses than 2A.

4. Plus or minus five db. were found to be the combined human error factor of observer and patient.

5. An average "quiet"—not sound-proof room was used for the tests.

CONCLUSIONS

1. Audiometers, despite the foregoing variations, furnish the best means of testing hearing acuity.

2. Any one of the four tested will be more useful in clinical practice than any other means now available for testing and recording hearing acuity.

3. The lack of purity of sound wave apparently did not introduce any serious errors that were not largely explainable by other causes.

4. A quiet room is essential. The need for a sound-proof room increases as the hearing loss to be tested decreases.

5. An audiometer should be part of the office equipment of every otologist.

Through the Otologists Committee on Audiometers and Hearing Aids,* the Council on Physical Therapy of the American Medical

*This Committee is composed of Drs. George M. Coates, Lee Wallace Dean, Edmund Prince Fowler, Austin A. Hayden (Chairman), Isaac H. Jones, Douglas MacFarlan, Burt R. Shurly, Horace Newhart and William P. Wherry. Mr. Howard A. Carter is Secretary. Several of the individual members have availed themselves of the services of such physicists as Bunch, Fletcher, Hartig, Knudson and Rogers.

Association places its facilities at the disposal of otologists for the purpose of correlating clinical experience, laboratory procedure and manufacturing detail. Acceptance by this Council should become an incentive to manufacturers to construct instruments in accordance with agreed upon clinical requirements and assure otologists that advertised claims meet required specifications.

DISCUSSION

MR. HOWARD A. CARTER, (by invitation), Secretary, Council on Physical Therapy, American Medical Association: As far as I am aware, this report is the first and only one in the literature on four audiometers tested concurrently. I have been asked to discuss this paper since I have had experience in this field with the Council on Physical Therapy, which has been aided by a group of consultants, all otologists, of which Dr. Hayden is Chairman. In the Journal of the American Medical Association, November 27, 1937, a report "Tentative Minimum Requirements for Acceptable Audiometers" was published. I have a reprint of that report in my hand and you may obtain one by writing to the American Medical Association. I should like to review some of the points considered when these requirements were formulated. It might be of interest for you to know that these requirements are the summation of experience of qualified men who have been working in this field for some time. Group opinion, at times, is difficult to deal with, but, when conclusions are reached, they are usually reliable. Even for the definition of the clinical audiometers difficulty was encountered. At the onset, the consultants had a long paragraph for the definition but when the group finished there was left but one short sentence, reading as follows: "A clinical audiometer is an instrument for measuring the acuity and range of hearing."

It was felt by the consultants that audiometers shall produce frequencies from 128 to 8192 cycles per second in sweep or in seven discrete frequency stops. The limits of the tolerable frequency variation are quite important and familiarity with this requirement might be most helpful to an otologist when salesmen come around with sales talks for audiometers. The frequency variation should not be more than plus five per cent at all frequencies. The audiometers shall be calibrated in decibels and should not be more than five decibels per step. The following range limits for intensity, according to the requirements, shall be plus one and one-half decibels per five decibels steps. The term "percentage hearing" was ruled as an inadequate method of expressing hearing loss.

Much discussion was devoted to the wave form. It was the opinion of some that the output should be a pure sine wave. Others believed that an absolute sine wave was not necessary. If the wave form did not follow the sine wave, some believed that harmonics were present. It was felt that harmonics of a low tone might be interpreted as a high frequency note to a person deafened to low tones but only moderately deaf to high tones. Dr. Hayden has apparently shown that wave form does not have as much to do with the successful operation of the instrument as might be expected.

The auditory chart or audiogram was considered by the consultants but no agreement was reached and no uniform chart recommended for adoption. It was felt that physicians may have adopted their own method of recording and might object to a uniform method of correlating data.

The definition of threshold of hearing adopted reads: "The threshold of hearing is the audiometer setting corresponding to the lowest intensity at which the person being tested is able to indicate correctly more than half the time that he is hearing."

The consultants recommended that the rules of the Council on Physical Therapy might well be adhered to by the manufacturers of acceptable audiometers so far as the marketing methods and advertising statements are concerned.

DR. T. C. GALLOWAY: What are the effects on audiometers of variations in voltage of the city circuit? Also, how may one know when a machine is out of order without testing it against some other machine? What are the other chances of an audiometer getting out of order that may go undetected?

MR. CARTER: Some audiometers are provided with voltage regulators which help maintain a constant intensity as predetermined by calibration.

Audiometers are on the market equipped with a sweep frequency and discrete frequency steps. In case of audiometers equipped with heterodyne circuit where one frequency beats against another frequency, unless provisions are made, the resultant output frequency of the unit will vary from time to time. Hence, to make sure that the unit is calibrated correctly, an indicating light is fixed on the panel of some of the units which lights when the dial is correctly set at a predetermined known frequency.

If the audiometer is out of order it is highly probable that an experienced operator would know it. However, in some instances,

there may be only a slight disturbance, for example, inaccuracy of calibration with slight irregularities, can be located only by examination and special tests.

DR. ALFRED LEWY: In the absence of Dr. Sonnenschein, I presume it devolves upon me to say something about audiometers and tuning forks. It is very refreshing to hear Dr. Hayden admit that audiometers vary. If he tried out several audiometers made by the Western Electric Company he would probably find that they also varied from each other. I agree that the audiometer is an adequate instrument for the measuring of the acuity of hearing. In addition it is of value in medicolegal cases, particularly in catching malingerers who feign a partial loss of hearing. It is practically impossible for them to give the same graph on repeated tests.

At the last meeting of the Triological Society, Dr. Ersner and a Mr. Podolsky, an acoustic engineer, presented a long and carefully prepared article on the results of their investigation of five makes of audiometers. Not one of them was accurate in all particulars. Every one had one or another fault which would disqualify it as an accurate scientific instrument. Moreover, while the audiometer measures the acuity of hearing, it is impossible to make a diagnosis in many instances, without a determination of the bone conduction. Otherwise the manufacturers would not have gone to the labor and expense of developing a bone conduction attachment and chart. They now admit this but did not at first.

In regard to tuning forks—when you are making a measurement of something that varies in caliber from time to time, as human hearing is known to do, you would not use a micrometer any more than an artisan does in measuring a piece of wood which is subject to change from moisture, temperature, etc. So I agree that for practical purposes it is not necessary to have the audiometer so accurately calibrated. Neither should it be offered to the profession as an instrument of scientific precision. I should also like to know why the prices of audiometers vary so greatly when for all purposes demanded by the standards set by the American Medical Association each instrument will give the same data. Why does one cost $200 and another $600?

I wish to call your attention to the fact that Professor Struycken, the inventor of the monochord, has brought out a tuning fork holder with a hammer activated by a spring which can be regulated to a standard intensity, so that the applied force can always be the same.

Tuning forks have been used for diagnostic purposes by several generations of otologists. We are familiar with their uses and their faults. I still believe that I can make a good diagnosis with tuning forks so far as pathology and indications for treatment are concerned.

DR. AUSTIN A. HAYDEN (closing): Regarding Dr. Galloway's question of the variation of 2048, I had an audiogram made in St. Louis on my own ears and on the instrument I think Dr. Galloway was talking about it showed a dip of only ten decibels. Just why that is I do not know. It goes without saying that there is a lot about audiometers that is not known as yet.

Regarding Dr. Lewy's question about the different makes of the same instrument giving different readings, it depends on what you mean by "different readings." I think most of them give rather constant trends in readings but they do not give the same readings. I do not think we could expect them to for there are so many variations in the air and in the noise level of rooms. I think many of these tests will be made in rooms that are not sound-proof and all of these are inescapable errors. As I read Dr. Ersner's article I was struck by the fact that Dr. Ersner and Dr. Lewy might have talked this over together. The trends in the audiometers are similar, they are not exact and I think are not expected to be exact.

About diagnosing lesions of pathologic conditions without bone conduction, no audiometer is put out now without bone conduction attachments. This addition was not made, I suspect, on account of the diagnosing of ear diseases but rather for the facility of fitting hearing aids where a bone conduction apparatus would work better than an air conduction apparatus.

About the cost of audiometers, Dr. Lewy wants to know why they are so expensive. I wanted to know that myself. I paid $640 for the 2A Western Electric audiometer I bought several years ago and am told that on every machine they sold they lost money. The manufacturers say that the variations depend upon the construction of the machine. They figure in the salary of a lot of men like Mr. Carter and they charge that to us when we buy an instrument.

I do not see any other way out for I thoroughly believe that this is the way hearing acuity will be measured in the future.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Meeting of Monday, January 3, 1938

THE PRESIDENT, DR. WALTER H. THEOBALD, IN THE CHAIR

Presentation of Cases

DR. J. R. LINDSAY

Five clinical cases were presented before dinner. Two of these, the cases showing visible movements of the stapes, and the case with cochlear function still present, and a return of facial movements, after the labyrinth operation (the canals and vestibule were opened posteriorly, and the promontory removed between the oval and round windows) and the decompression of the facial canal, will not be discussed unless questions are asked.

The two cured petrositis cases will be presented later.

The fifth case—presented as "laryngeal vertigo" has been ill for eight months. In May he began to have severe coughing spells and during the more severe attacks of coughing he falls down unconscious, and in fifteen or twenty seconds gets up again, apparently quite normal. He broke his fibula in one fall.

He has been observed on some of these occasions, and at least on one occasion had epileptiform movements.

He only falls down when the coughing spell is severe. At one time he would fall as often as twenty times a day, but in recent months the attacks have become infrequent. Neither the local examination nor the general physical or neurologic examinations have revealed the cause of his coughing spells or the falling. The attacks could not be induced by breathing carbon dioxide, rebreathing or pressure on the carotid sinus.

The name "laryngeal vertigo" is used here because that term has been used for such cases in the literature. There is no vertigo.

Attention is drawn to an article by Dr. J. Gordon Wilson in the Archives of Otolaryngology several years ago pointing out the similarity of laryngeal vertigo to cataplexy.

DR. S. A. FRIEDBERG: I think Dr. Galloway's modesty kept him from talking about these cases. I will briefly outline the histories.

Dr. Galloway was the surgeon in each instance and his judgment was undoubtedly responsible for the excellent results. The first case was that of an eleven-year-old boy who had a complete mastoidectomy after five weeks of persistent suppurative otitis media. He was discharged practically healed in ten days. He returned a week later with profuse recurrent discharge from the middle ear and complaining of severe left-sided headache and pain around the eye. Temperature had been 101° F. each evening. Roentgenogram showed some involvement of the petrous apex. The suppuration was mostly from the mastoid wound. The wound was reopened; an area of softened bone was found behind the posterior semicircular canal; this was followed towards the petrosa and frank pus was encountered. He made a complete recovery. The operation was performed on the basis of severe headache, pain in the eye, elevated temperature, recurrence of drainage from the mastoid wound and x-ray findings.

The second case was very complicated. The boy was brought into the Cook County Hospital in diabetic coma with a history of having had a tooth extracted two weeks previously. There were frank signs of meningitis; he had paralysis of the second through the eighth cranial nerves on the right side. There were signs of diabetic acidosis in addition to the basilar meningitis. He began to recover clinically with control of the acidosis. In the course of five or six weeks sequestration of the hard palate and part of the sphenoid took place. After approximately five weeks he complained of excruciating headache and pain in the eye on the right side. We felt there was some involvement of the petrous apex, probably secondary to osteomyelitis of the sphenoid bone. Dr. Galloway explored the mastoid and encounterd a large abscess in the anterior petrosa. The boy was relieved of headache. At the present time there is a large defect in the palatospenoidal area. The diabetes is controlled. There is a fistula into the floor of the sphenoid from the petrous apex demonstrable by alcohol irrigation via the middle ear. The status of the involved nerves remains the same. There is slight drainage from the middle ear. Carotid ligation was considered because of the proximity of this vessel to the large sphenoidal defect, but so far he has progressed satisfactorily and the lesion is quite clean.

Dr. T. C. Galloway: With reference to the second case mentioned by Dr. Friedberg, we thought this might be an osteomyelitis of the petrosa arising by direct extension from the nasopharynx for several reasons. In the first place, the third, fourth, fifth, sixth and seventh nerves were already involved before the ear had drained. The palate was first involved. At operation it was significant that

the posterior cells of the mastoid were nearly normal, the anterior-superior group more involved. The petrosa was found to be a little more than a shell filled with thick pus. The process may have gone up through the tube, but quite likely the areas of sequestration represent the true pathway.

Suppuration of the Petrous Pyramid

DR. J. R. LINDSAY

(Author's Abstract)

A presentation of pathologic material from a group of meningitis cases, of otitic origin, and a report of three proven clinical cases of petrous apex suppuration, successfully treated.

The pathologic presentation included photomicrographs from seven cases of petrous pyramid suppuration and one case of labyrinthitis with abscess of the saccus endolymphaticus, which were collected over a period of seven years. These eight cases were obtained from a group of twenty-eight cases of meningitis of various types, of which a total of fifteen had an associated otitis media.

Some of the important features illustrated by the pathologic examination were as follows:

Macroscopic findings at autopsy usually did not give sufficient information to indicate the pathway of extension to the meninges. In order to get the exact information which is necessary if an adequate method of treatment is to be arrived at, the histopathologic examination is essential.

Out of seven cases of abscess (including three clinical cases) in the petrous apex, five had no fistula to the middle ear or mastoid, while in two cases a microscopic tract of suppuration was demonstrable. These cases show that the abscess in the apex frequently cannot be reached from the mastoid, that it is frequently, if not usually, of the closed type, and that it may rupture in any one of several directions. The infecting organism was hemolytic streptococcus in most cases, but three cases of pneumococcus type III were found and one of pneumococcus type I. In one of the type III pneumococcus cases there was a large abscess in the apex, with rupture of the middle fossa and meningitis within two weeks of the onset.

In the other pneumococcus cases the disease was more prolonged, and accompanied by little fever until the meningeal infection developed.

In the streptococcus cases the course was sometimes prolonged, with little or no fever, and in other instances there was a stormy febrile course from the onset. A septic temperature occurred only in the streptococcus cases, and usually indicated a venous sinus thrombosis. In suppuration of the apex there may be involvement of the carotid venous plexus, the cavernous and petrosal sinuses before the jugular bulb or lateral sinus are involved. The most essential feature of the treatment in such cases is the drainage of the suppurating focus in the bone.

In every case the infection had progressed into the pyramid by way of pneumatic spaces, with production of an area of suppuration and bone absorption, which later ruptured through the cortex to the meninges. The bone marrow showed marked resistance to the infection. A diffuse fibrosis occurred in the marrow spaces in most cases, affecting gray and red marrow alike, with some osteogenesis adjacent to the suppurating focus. This reaction in the marrow is not true osteomyelitis, and provides an adequate defense against invasion and destruction by the infection.

Bilateral acute osteomyelitis occurred in one case as a terminal process.

The most constant symptom in the author's group of cases was pain, the location of which, in the early stages, was an important indication as to the site of the lesion. Aural discharge was absent in two; sixth nerve palsy was present in three of the apicitis cases. A positive Babinsky's sign on the opposite side was noted as an early sign in two recent cured cases, indicating that a local meningeal reaction was present in the middle fossa, although the spinal fluid was normal.

The rœntgenographic findings are a valuable aid to diagnosis. The axial views have been most useful in the author's experience, although the occiput down and Stenver's views are routinely used.

These cases demonstrate that petrous pyramid suppuration should be diagnosed while the infection is still in the intrapetrosal stage. A safe means of procuring drainage from a "closed" abscess is a necessity. If surgery is delayed until rupture of the cortex takes place, a meningitis may be unpreventable. The intra-petrosal method of draining an abscess in the apex from the mouth of the Eustachian tube was used by the author in four cases with a satisfactory result.

DISCUSSION

DR. E. W. HAGENS: About the only way I can discuss this paper is to compare and contrast Dr. Lindsay's experiences with my own. His presentation is difficult to discuss because his findings speak for themselves. I think we all appreciate that the petrous bone varies a great deal in its anatomy. In the newborn it consists of an outer layer, the periosteal bone; an inner layer, the endosteal; and in between a layer which consists of a great deal of marrow. That marrow layer changes greatly as time goes on. In some instances the spaces become smaller and the bone becomes solid. In other cases considerable marrow will remain, particularly in the apex. In still other cases pneumatization takes place as in the mastoid and we may have a variety of pictures of the petrous bone anywhere from a solid petrous bone to extremely pneumatized bone. I think that must be borne in mind to evaluate clinical cases. I followed a number of children at County Hospital from six months to fourteen years of age. Twelve died with meningitis. I did not know whether they had petrositis or not, but they had otitis media. They had meningitis and died. I thought it would be interesting to know if the petrous bone was involved, because clinically one has to decide such a matter before death—whether we should operate or not—how we should operate—how conservatively or how radically. Six of the twelve were operated upon, the others were not. Then the question was: Had we missed any of those cases? Had it been a petrositis and did it break through and cause meningitis?

My experience differs somewhat from Dr. Lindsay's seven cases, but if you include the others to make 15 my figures would coincide better with his. It seems to me that infection spreads mainly by the pneumatized spaces, then by marrow, and last of all it will erode bone. Hard petrous bone, however, will resist infection. I have found osteomyelitis in the petrous apex in three cases—a large erosion so that you could hardly recognize it as a marrow cavity. The age of these children was three, four, and five years respectively. It has often been stated that, given a certain case where there is little or no pus found, we are dealing with a thrombotic phenomena, and by extension through the thrombosed vessels meningitis develops. I do know that that can happen, but I did not see it in the twelve cases I examined. In one the pathologist was sure he would find it, because there were thrombi throughout the body. Yet the sections did not reveal any thrombi. I am speaking only of the petrous bone. I also found labyrinthitis in most of these cases. It seemed to be secondary to the meningitis. In only one case could I find a spread

through the round window, and here no perforation was seen, just a thinning of the membrane of the window. I believe the labyrinth is well insulated, so that infection does not travel as readily as you might think.

Regarding bacteriology, Dr. Lindsay mentioned that in some of his cases he found streptococcus. In my series there was streptococcus in four, pneumococcus in six, and one definitely a pneumococcus type III, influenza in one and a mixed infection in another. We have talked since 1918 about influenzal otitis media. It was interesting to me to see hemorrhage through the labyrinth—an entirely different picture from the other bones, and it looks as if in influenza we get a different picture throughout the ear. I have enjoyed Dr. Lindsay's paper, and I think with a summation of all the series along this line, we will finally appreciate the variations possible in petrositis and be able to follow these cases through more intelligently and treat them surgically.

Dr. W. E. Grove: I have had considerable trouble in getting rœntgenograms of this type, and I would like to know how Dr. Lindsay produced the excellent pictures he showed.

Dr. Samuel Pearlman: I want to congratulate Dr. Lindsay on his fine paper. It is admirable in all respects. I could not help thinking as he spoke how much there remains, for me at least, before the picture of petrositis will be altogether clear.

My memory goes back to a case of some years before which illustrates, I think, the general attitude of that time. A youngster had an otitis media for over two weeks and began to complain of terrific headache. Impelled by a fear of impending meningitis, he was operated upon and within a day or two developed an external rectus paralysis. I remember how happy I was then because we felt that our troubles would now be over. We felt that it was, in the parlance of the day, a Gradenigo's syndrome occurring in the presence of an otitis media, and we all know that the largest majority of these patients get well without any further interference. We also knew that a certain number of them developed meningitis but we thought there was not much we could do to prevent that.

On the other hand, a few weeks ago on our service at the Cook County Hospital, a youngster was entered with a picture of a frank meningitis. Hemolytic streptococcus was recovered from the spinal fluid. He had a unilateral otitis media of about eight days' standing with an intact membrana tympani, and a paracentesis was done at once. There had been no discharge prior to this. Sulfani-

lamide therapy was employed and the child rapidly improved. The mastoid process was not operated upon. In former days it would have been the one thing there was to do, along with spinal fluid drainage. The new chemotherapy is so markedly changing our attitude in these cases that it seems that we are facing what may be a rather important revision of our operative indications, particularly with regard to otitis media complicated by meningitis. Mastoiditis, of course, has indications all its own. Furthermore, one cannot help speculating about the pathology of these troublesome cases and the nature of a process which yields so quickly to drug therapy. Surely bone destructive lesions cannot heal in 48 to 72 hours. We may look forward, I hope, with a great deal of optimism and with expectation to a rapid disappearance of the confusion of today, based on such works as we have heard Dr. Lindsay report.

DR. T. C. GALLOWAY: I want to point out one value of Dr. Lindsay's work. He has visualized the pathology. He has pointed out the proper approach in each case, and I think if we follow that up we are going to make, in many cases, not only a positive diagnosis of petrositis, but are going to be able to make a specific diagnosis of the part of the petrous that is involved, and therefore get indications for our operative procedure.

DR. JOSEPH C. BECK: As an oldtimer, with considerable experience in operating on mastoids in public institutions and private practice, up to this day I have not done an operation on the petrous pyramid. I recall very few cases of meningitis following mastoid operation, and when this work on the petrous pyramid was first brought out and surgery enthusiastically spoken of, I was very unhappy about the whole thing and said to myself—"now we will have a holocaust of deaths from this enthusiasm." In going through the literature and checking the cases reported by Kopetzky and other men, including two cases operated upon successfully by my associate, Dr. M. R. Guttman, I am glad to say that I have lived enough to change my mind. As Dr. Pearlman and Dr. Galloway mentioned, both the gentlemen who presented the paper and the discussors rounded out the whole picture fairly well. Only time will tell exactly where we stand on this situation of when and how to operate. Just because a man has not seen any deaths from meningitis following mastoid operation is not a criterion. There are many cases on record which had been diagnosed properly and operated upon in the proper way. I do not think of the boring operation when I say this, but I think of the uncapping of the petrous pyramid through an opening that you can see at the base of the skull. Some of the cases we

heard reported here showed very clearly that somebody was just too late in letting this abscess formation go on through an unprotected meningeal membrane. Fortunately they are not all the same. These people would have died had the doctor not operated. I congratulate the Society on this presentation and I am sure you will go away with greater enthusiasm to study the subject and not allow meningitis to develop.

DR. J. R. LINDSAY, (closing): I want to thank Dr. Hagens, Dr. Pearlman, Dr. Galloway and Dr. Beck for their kind discussions.

In answer to Dr. Grove's question, I have shown you here only one view of the petrous pyramids, the so-called axial view. We also use two other views, the occiput down view, and Stenver's views. The latter view has been favored in Europe, but in our experience the axial view has been more useful. The three views are taken in each case. If there is erosion of the superior angle the Stenver's or occiput down views might show it best. On the other hand, suppuration in the lower part of the apex, or confined within the apex, shows best in the axial view.

I have avoided discussing the use of sulfanilamide purposely. We have had some experience with it during the past year, but not enough to draw any conclusions. It may be said that our experience with this drug to date has not indicated that any of the well established surgical principles may be discarded.

This presentation has included only proven cases of petrous pyramid suppuration, eight of them autopsied cases. During the eight year period in which they were collected other cases of petrositis have also been seen; and have terminated fatally. I have included all cases in which the necessary postmortem examination was possible.

Studies of Immunization of the Upper Respiratory Tract

T. E. WALSH, M.D. (by invitation).

(Author's abstract)

The normal defenses of the upper respiratory tract have been emphasized recently by Proetz and others who have stressed the importance of the mucous blanket and the ciliary activity as a first line of defense against infection. Various factors, both physical and dietary, combine to break down this first line of defense. Dr. Cannon and I were interested in ascertaining whether an adequate second line of defense could be obtained against invasion of micro-organisms.

The results of subcutaneous vaccination against colds has been on the whole disappointing. The ideal conditions for tissue immunity are said to consist of an accumulation of phagocytic cells and antibodies in the tissues. These conditions can be obtained by the injection of vaccine into such tissues. This has been shown by Cannon and others. We were interested in ascertaining whether this could be accomplished by vaccination of the nasal mucosa. Experiments are described which demonstrate that following the application of vaccine of Paratyphosum B in the nose the antibody titer of the nasal musosa is higher than that in the mucosa following subcutaneous injection of the same antigen. Histologic examination of the tissue reveals a marked accumulation of large monocytes in the subepithelial tissues. Further experiments are described in which two different antigens were given to the same animal, one by means of intranasal application, the other by intraperitoneal injection, and the tissues and serum of the animal were titrated simultaneously against both antigens. It was found that where the serum titer is the same in both antigens the tissue titer of the mucosa and lungs is constantly higher for the antigen given intranasally and that when the serum titer to the "general" antigen is many times higher than that to the "local" antigen the respiratory tissue titer is the same for both antigens. The explanation is offered that there is actually local formation of antibodies in the respiratory tissues following local vaccination. The conditions in the tissues following local vaccination therefore are those which are said to be ideal for tissue immunity.

Another series of experiments in which animals were injected with a virulent organism are described, and it is seen that following intranasal vaccination animals were immunized to several hundred lethal doses of the organism given intranasally. It is pointed out that the probable pathway of infection following intranasal application of living virulent organism is through the lungs. Material put in the nose is aspirated directly to the lungs. It is shown however that in animals under anesthesia with a tracheotomy in which the upper end of the trachea is firmly plugged, virulent organisms do pass through the nasal mucous membrane and enter the blood stream in unimmunized animals, whereas similarly treated animals that had been vaccinated intranasally for five days longer were protected. The ease with which substances put in the nose are recovered from the lungs led to experiments on the effect of various intranasal medication on the lungs of rabbits. Slides are shown illustrating the findings.

The clinical application of the experimental work described was in prophylaxis against the common cold. A polyvalent vaccine was used as a nasal spray with encouraging results—some 80 per cent of the patients so treated being free from colds—whereas in the control group there was a variation of colds in 50 per cent of the patients.

DISCUSSION

DR. FRANK J. NOVAK, JR.: Dr. Walsh's paper is a consolidated report on work carried on over a considerable period of time. This report is both interesting and important. It is interesting from a purely academic standpoint as a piece of excellent investigation, and important because of the clinical and practical implications which it carries. I believe that the proper approach to the general problem of upper respiratory infections is from an immunologic standpoint. We have known from the work of Cannon and others that it is possible to produce local tissue immunity in the skin. Upon injection of antigen into the skin it is possible to measure accurately the humoral response of the skin. Moreover, it is possible to measure the cellular response to that injection. We know that the antibody titer of that piece of tissue is increased for the time being. We also know there is a mobilization of various cells that have to do with the mechanism of immunity.

Applying that idea to the nasal mucosa we have seen the result. I think Dr. Walsh has perhaps investigated the humoral response, that is the antibody response, rather than the cellular. I like to look upon the thing as being particularly a cellular and secondarily a humoral response.

If we drop a solution containing streptococcus into the nose of a rabbit or human, we can expect to find a response to the antigen in the formation of a specific antibody. If we drop a pneumococcus into the nose we can expect a specific response. Practically, when we are attempting in the office or in the clinic to immunize against the organisms which are causing head colds it is not sufficient to take one type of streptococcus or pneumococcus or catarrhalis or staphylococcus, and expect any kind of clinical result. In other words, the whole thing points away from the use of autogenous vaccines. Polyvalent vaccines containing all kinds of organisms are necessary if we attempt immunization.

I was struck with one fact in particular, that is, while the application of a vaccine directly to the nasal mucosa produces a measureable rise in antibody content of that mucosa, the injection over a long period of time will do the same thing, but to a lesser extent.

It seems that if we are to be practical about it, it might not be a bad idea to inject the vaccine and get a blood response, to apply it locally to the throat and nose and get a local response. I would like to ask Dr. Walsh whether anyone would have the hardihood to try an injection into the nasal mucosa with a moderate amount of vaccine. We know the response of the skin to injections is greater than the response to surface application. If we remove the horny layer of the skin of the arm or belly by abrasion, then apply to that skin a compress of filtrate or vaccine, we will get a definite change in tissue which is directly measureable both by antibodies and by mobilization of cells that have to do with immunity, but I think we get a greater response if we inject into the skin than if we apply on the outside, and so I wonder if it would not be feasible to inject the mucosa rather than to try to raise the level of immunity by mere topical application of antigen.

I was very much interested, and want to compliment Dr. Walsh for a very fine piece of work.

Dr. George Shambaugh, Jr.: I have used the method of intranasal vaccination against colds for two years. While I have not, as yet, tabulated the results, I have been impressed with the good results and am using the method with increasing frequency in people who are subject to frequent colds, and particularly to recurring attacks of sinus infections. I believe that this method is a real advance in the prevention of colds.

Dr. O. E. Van Alyea: It seems unusual to hear a paper on treatment of colds by vaccines, or any paper on nasal therapy, without mention of the virus which is supposed to be the initial invader in nasal infections. I wonder if Dr. Walsh has considered this virus in his study. Dr. Walsh's work combined with the investigations which are being carried on by Fenton and Larsell and others should be of great value eventually. Whether or not we can apply it clinically as yet is doubtful. As the doctor stated, it is difficult to obtain authentic reports from clinical patients. Another factor is the season variability. I think Dr. Shambaugh's results this season may be due to the fact that we have not yet been exposed to a severe cold epidemic. So far this season we seem to have avoided such an epidemic.

Dr. Alfred Lewy: I would like to ask Dr. Walsh if he noticed in his figures that the titer of the antibodies in the lung was as high or frequently higher than in the nasal mucosa, and how he accounts for that. There is a much higher epithelial surface in proportion in

the lung than in the nose. Does he think that the respiratory tract reacts as a whole, or is it a purely local phenomenon? Is the vaccine available for experimental purposes, and what is the cost?

Dr. T. E. Walsh, (closing): First of all I want to thank Dr. Novak for his able discussion of my paper. It has been my impression, and I believe that of other investigators, that antibodies are actually elaborated by histocytes. An area of histocytic infiltration is, I believe, necessary before vaccination will be effective. I think this is demonstrated in our experiments. I agree with Dr. Novak's remarks regarding polyvalent vaccines. We controlled the specificity of the antibodies in our experiments by duplicate titrations with the homologous organisms and a control. Where Paratyphosum B was used in the vaccine we controlled the titrations with B. typhosum and there was absolutely no agglutination of the control organism. I think it would be somewhat heroic to inject vaccine into the nasal mucosa; as a matter of fact we did this in one patient and he had a very marked reaction although a very small dose was injected into the inferior turbinate. He complained of severe headache and malaise and had a fever of 104 F. I believe the injection is probably actually intravenous as undoubtedly the large blood spaces in the turbinates would be entered. I do not know just how the vaccine in an intranasal spray gets through the epithelium and into the subepithelial tissue.

Dr. Van Alyea spoke about the virus of colds. It is true that the investigations of Dochez have shown that a virus may be the causative factor in colds. I believe its action is either to lower the resistance of the tissues or to enhance the virulence of the invading organisms which produce the symptoms of the common cold. The experimental work in the transmission of colds with the virus has shown that this is possible in only 30 or 32 per cent of cases. The etiologic factors in the other 70 per cent are those which we discussed in our opening paragraphs.

If the immunologic reaction is so specific I think it is important to have many strains of each organism used in the vaccines. In answer to Dr. Lewy, the lung titers in our animals were as high or higher than those of the nasal mucosa. This is undoubtedly because large amounts of vaccine are aspirated directly into the lungs from the nose and antibodies are presumably elaborated by the alveolar macrophages.

Abstracts of Current Articles

NOSE

Giant Osteoma of the Right Frontal Sinus With Invasion of the Orbit. (Osteoma gigante del seno frontale di destra con invasione dell'orbita).

Pieri, P. F., Valsalva, 13:119 (March), 1937.

The rarity of a case of this type makes it worth recording. This enormous osteoma, weighing about 105 grams when completely removed, occupied practically the entire cavity of an exceptionally large right frontal sinus, a portion of the ethmoidal sinuses and the orbital chamber.

The patient gave a history of falling from a carriage seven or eight years previous and striking the right frontal region of his head. Shortly afterward a heavy sensation appeared in that area and later it became a true and constant pain. The author considers this injury to be the stimulating factor which created reactive processes of the embryonic periosteal and connective tissue germinal centers of the part involved. This statement is confirmed by the histopathological study of the tumor mass.

SCIARRETTA.

Injuries of the Frontal and Ethmoidal Sinuses with Special Reference to Cerebrospinal Rhinorrhea and Aeroceles

Cairns, Hugh (London), J. Laryng. and Otol., 52:589 (Sept.), 1937.

The author shows in this article the various ways in which cerebrospinal rhinorrhea and intracranial infection may occur after fractures of the frontal and ethmoidal sinuses. The cases, made up largely of motor and airplane accidents, are usually injuries produced by violent impacts on the forehead or face. In the more severe accidents, the upper and lower jaws are also fractured and there is dissolution of the bony connections of the facial bones to the base of the skull.

In fractures of the frontal sinus and the cribriform plate, risk of intracranial infection is not necessarily immediate but may occur at a later date with any new septic process in the nose. Leptomeningitis is the usual result but brain abscess may occur.

The author advocates repair of the injured dura by means of transfrontal operation and sutures or fascial grafts to reduce this risk.

He feels that greater accuracy of diagnosis of the site of injury to the cranial base is necessary before immediate operation with the attendant risk of aggravating shock already present can be justified. He advocates close collaboration "between those who work below and those who work above the cribriform plate."

PHARYNX

Hairy Polyp of the Epipharynx (Polipo peloso dell'epifaringe).

Di Vestea, D. (Univ. of Rome), Valsalva, 13:153 (April), 1937.

A report of a case of congenital hairy polyp removed from the posterior-superior surface of the soft palate on the right side is presented.

The author reviews the literature extensively on the histopathological classification of these types of tumors and places this growth in the third and fourth group of Schwalbe's tabulation.

The tumor removed consisted of tissue derived from the three embryonal germinal layers. It is interesting to note that this tumor was covered partially by mucous membrane, which in some areas was squamous stratified epithelium; in others, cuboidal and cylindrical ciliated epithelium. Other portions of the polyp were covered by the typical dermal layer, containing hair, sudoriferous and sebacious glands. In its center there was a large blood vessel and a piece of formed cartilage covered by perichondrium. Smooth muscle tissue and glandular masses resembling salivary glands were also present.

Therefore, considering these histologic findings, he concluded that the tumor, while it was not a true teratoma, may more logically be called an epignathus.

The article contained nine photomicrographs.

SCIARRETTA.

Erosion of the Large Blood Vessels of the Neck in Peripharyngeal Suppurations. (L'erosione dei grossi vasi del collo nelle suppurazioni perifaringee. Considerazioni sui casi raccolti nella letteratura e su 3 osservasioni personali).

Traina, S. (Univ. of Pisa), Valsalva, 13:105 (March), 1937.

Cases reported in the special literature up to 1935 are discussed. The author considers the report of Salinger and Perlman of 1933 to contain the largest collected number, 227 cases, and to these he adds 33 more cases which the American authors had missed during their search. He adds three case reports of his own, presents three statistical tables, and mentions Tsoukermann's article of 1935.

Traina believes that severe hemorrhages due to peripharyngeal suppurations are more frequent than is commonly thought and, while the general literature seldom reports such cases, the literature of the specialty records approximately 300 cases.

He states that the mortality is higher from hemorrhage due to abscess located in the retro-latero-pharyngeal region where the internal carotid is most frequently involved than to peritonsillar abscess where the external carotid and its branches are perhaps more apt to be injured. The mortality is also without doubt much higher in cases where ligation of the vessels was not attempted.

In considering the etiological factors of such a high mortality, the virulence and nature of the germ is of primary importance; the incomplete and delayed intervention is secondary; then, the degree of local and general resistance; and lastly, the arterial anomalies.

The repeated and small hemorrhages are generally a warning of an impending severe hemorrhage, therefore, the surgeon should be prepared to ligate quickly and effectively one or more blood vessels.

SCIARRETTA.

LARYNX

Treatment of Simple Chronic Laryngitis.

Clerf, Louis H. (Philadelphia), Arch. Otolaryng., 27:261 (March), 1938.

To summarize briefly, chronic laryngitis is a nonspecific inflammatory disease, the causes of which are many and varied. Treatment consists of employment of local measures and removal of the cause. Although the effects of local treatment are temporary, such treatment is important. Etiologic factors must be sought for in the patient's habits, occupation, environment and general health. Improper use of the voice, repeated attacks of acute laryngitis and disorders of the accessory nasal sinuses, the nose and the oropharynx are the most important of these factors. The results of treatment are dependent on the finding of the cause and on its correction or removal.

TOBEY.

EAR

Otitic Sinus Thrombosis.

Sutherland, J. M. (Detroit), Arch. Otolaryng., 27:1 (Jan.), 1938.

From a comprehensive review of the literature based on the physiologic, anatomic and pathologic conditions involved in the causation, production and distribution of suspected thrombosis of

otitic origin and from the study of the records of past clinical experiences, it appears that no unified surgical procedure can be adapted for this condition other than the eradication and drainage of the primarily infected area. The use of surgical intervention on the jugular vein is still controversial.

While great advances have been made in the diagnosis and treatment of sinus thrombosis, much still remains to be desired in treatment in accomplishing a complete eradication of the infective foci.

TOBEY.

Treatment of Purulent Otogenic Meningitis (Sulla cura della meningite purulenta otogena).

Torrini, U. L. (Firenze), Arch. Ital. di Otol. Rino and Laring., 49:133 (March), 1937.

The interest shown by the writer in the cure of this pathologic condition has given him the opportunity to treat a considerable number of these cases. He has tried many and various methods.

In 1935 he reported two cases of septic meningitis which were treated and cured by intermittent drainage of spinal fluid and injection of colloidal gold. Considering the possibility of falling in the category of spontaneous cures, as believed by various authors, he could not draw any definite conclusion from the two cases at this time.

During 1936 he obtained another cure. This patient, a young person, had a right suppurative mastoiditis complicated by meningitis and labyrinthitis. Colloidal gold was injected into the arachnoid space immediately after the operation. This medication was given daily, with improving clinical symptoms and laboratory findings. When the autogenous vaccine was administered daily in increasing doses, all symptoms were aggravated and the spinal fluid gradually became turbid. The nonspecific protein therapy was started again and the patient manifested improvement until completely cured.

It is interesting to note that the vaccine aggravated the condition instead of improving it. The author attributes the unfavorable results to the toxic effect of the vaccine on the arachnoidal tissue, which fact has been confirmed by other observers.

Torrini has treated many cases of purulent meningitis of otogenic and rhinogenic origin with colloidal gold. Practically all cases showed improvement under such therapy, but the patients were removed from the hospital, refusing further care, and consequently terminated fatally.

SCIARRETTA.

Are Irrigations Injurious or Dangerous or Recommended in Purulent Otitis Media? (Sono pericolose, o dannose, o da raccomandarsi le irrigazioni nelle otiti medie purulente?)

Salaris, E. (Univ. of Sassari), Arch. di Otol. Rino and Laring., 49:115 (March), 1937.

Purulent otitis media was treated by irrigation for a considerable number of years, until Gradenigo called attention to the danger of this procedure. He recommended the dry or semi-dry method which was immediately indorsed by the German school and now is universally employed as the approved form of therapy.

Salaris argues emphatically and extensively in favor of irrigation in otitis media, and advises sterile or mild medicated solution in both acute and chronic cases. With this method the pus is removed quickly and effectively from the external auditory canal and tympanic cavity; therefore, if instillations of medicated fluids are prescribed they will be more beneficial. Excluding cases with cholesteatoma, he considers the dry or semi-dry therapy insufficient and irrational, as the arguments for these methods are based only on theories and not on practical and scientific facts.

He quotes David Shapiro's article reported in the Archives of Otolaryngology, in which the author comments on five cases treated by irrigation of the middle ear with a pint of 1 per cent methylene blue solution one hour prior to operation. Another 50 cc. of the same solution was injected forcefully into the ear just before making the primary incision and no trace of the dye could be found in the antrum or mastoid cells.

The author reports experiments conducted on twelve patients on whom he had made a count of the bacteria and of colonies grown from pus obtained from the auditory canal and tympanic cavity before the ear was cleaned, after dry treatment, and after irrigation. He found considerable reduction in the number of bacteria and colonies grown from material collected after irrigations. These findings confirm those of Calicetti reported in 1917 on the bacterial count only.

SCIARRETTA.

Treatment of Recurrent Vertigo (Menière's Syndrome) by Subtemporal Destruction of the Labyrinth.

Putnam, Tracy J. (Boston), Arch. Otolaryng., 27:161 (Feb.), 1938.

An operation for the relief of recurrent vertigo is described. This operation consists in destruction of the vestibular ganglion through an opening into the superior canal from the upper anterior

surface of the petrous ridge, approached by a small subtemporal decompression.

Five operations in two cases are reported; in each case the vertigo was relieved while hearing was preserved. In one of these there was no evidence that an infection of the ear had ever existed; in the other, there had been an infection on one side, but both sides had to be operated upon before relief was obtained.

TOBEY.

The Anaphylaxis Reaction: Tissue Changes as Observed in Living Tissue in the Rabbit.

Schenck, Harry P. (Philadelphia), Trans. Am. Laryng. Assoc., 1938.

The experiments demonstrate by direct microscopic observation certain arteriolar changes in the moat chamber in the rabbit's ear. In the actively sensitized animal, these changes follow intravenous injection or introduction of the anaphylactogen into the moat.

MISCELLANEOUS

Differential Diagnosis Between Thrombosis of the Lateral Sinus and Acute Bacterial Endocarditis.

Rosenwasser, Harry (New York). Arch. Otolaryng., 26:668 (Dec.), 1937.

Certain diagnostic features which aided in making the correct differential diagnosis between thrombosis of the lateral sinus and acute bacterial endocarditis have been stressed. When the Ottenberg differential blood culture indicates large numbers of colonies in the cultures of blood from the jugular vein and a peripheral vein in the arm, it points to endocarditis as the cause, whereas a large number of colonies in one or both jugular veins and many fewer colonies in the arm point to sinus thrombosis.

Correlation of the bacteriologic characteristics of the aural discharge and the pus from the mastoid with blood cultures, as described by Libman and Celler, Ottenberg and Friesner, is helpful. Important in the differential diagnosis is the fact that none of the pneumococci, with the exception of the type III pneumococcus, cause sinus thrombosis. The significance of the embolic phenomena should be emphasized again because of their diagnostic importance. Cutaneous embolic lesions, petechiae, have never been observed by me in a case of sinus thrombosis uncomplicated by bacterial endocarditis. Changes of the fundi, varying from slight blurring of the margins of the disks to four diopters of papilledema, occurred in 16 per cent of the cases of sinus thrombosis which

the author and his associates have observed. It is uncommon in a case of acute endocarditis to note any change in the fundi other than embolic lesions or their manifestations, namely, petechiae, Roth spots or Doherty-Trubeck lesions. The changing character of the cardiac murmur or murmurs from day to day is significant of endocardial involvement. A definite history of aural disease, mastoiditis and a tender gland at the angle of the jaw, associated with corroborative local evidences of venous involvement, are additional factors in determining the correct diagnosis.

In conclusion, it must be apparent that these cases of serious borderline conditions require the closest co-operation between the otologist, the internist and the bacteriologist for their proper solution.

TOBEY.